SAFETY AND EFFICACY OF LOW DOSE MAGNESIUM SULPHATE REGIMEN IN ANTEPARTUM ECLAMPSIA AND A COMPARATIVE STUDY OF DHAKA REGIMEN WITH PRITCHARD REGIMEN IN ANTEPARTUM ECLAMPSIA

Dissertation submitted to

The Tamilnadu Dr. M.G.R. Medical University
in partial fulfillment of the regulations
for the award of the degree of

M.D. – Branch II
OBSTETRICS AND GYNAECOLOGY

K.A.P.Viswanathan Government Medical College
Tiruchirappalli

The Tamilnadu Dr. M.G.R. Medical University

Chennai

March - 2010

CERTIFICATE

This is to certify that the dissertation entitled "SAFETY AND EFFICACY OF LOW DOSE MAGNESIUM SULPHATE REGIMEN IN ANTEPARTUM ECLAMPSIA AND A COMPARATIVE STUDY OF DHAKA REGIMEN WITH PRITCHARD REGIMEN IN ANTEPARTUM ECLAMPSIA" is a bonafide work done by **Dr. M. NAGAMANI** at K.A.P.Viswanathan Government Medical College, Trichy. This dissertation is submitted to Tamilnadu Dr. M.G.R. Medical University in partial fulfillment of University rules and regulations for the award of M.D. degree in Obstetrics and Gynaecology.

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DECLARATION

I **Dr. M. NAGAMANI**, solemnly declare that the dissertation titled, "SAFETY AND EFFICACY OF LOW DOSE MAGNESIUM SULPHATE REGIMEN IN ANTEPARTUM ECLAMPSIA AND A COMPARATIVE STUDY OF DHAKA REGIMEN WITH PRITCHARD REGIMEN IN ANTEPARTUM ECLAMPSIA" is a bonafide work done by me at K.A.P.V. Government Medical College, Trichy, during 2008 - 2009 under the guidance and supervision of Prof. Dr. PREMAVATHY PRABHU ELANGO, M.D., DGO., Professor and Head of the department, Obstetrics and Gynaecology. This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University, in partial fulfillment of University rules and regulations for the award of M.D. Degree (Branch – II) in Obstetrics and Gynaecology.

Place: Trichy
Date : **Dr. M. NAGAMANI**

ACKNOWLEDGEMENT

I am extremely thankful to the **Dean Dr. N. Balasubramanian, MD.,DD., K.A.P.V. Government Medical College**, **Tiruchirappalli** for granting me permission to undertake the study and to avail the facilities needed for my dissertation work.

I have a deep sense of gratitude to the **Prof. Dr. Premavathy Prabhu Elango, MD.,DGO.,** ***Professor and Head of the department,*** Obstetrics and Gynaecology who gave immense support and encouragement and all the facilities to complete this work.

I have profound happiness in recording my gratitude to the **Associate Prof. Dr. Rani, MD.,DGO.,** for being a great source of encouragement in shaping this report.

I am immensely happy to thank my guide **Dr. Bama Ramesh, MD.,DGO.,** for her invaluable help and expert guidance during this study.

I am extremely thankful to **Dr. Vidhya Ravi, MD.,DGO., Dr. Prasanna Lakshmi, MD(OG).,** Assistant Professors of Obstetrics and Gynaecology for their unlimited encouragement, guidance and help during this study.

I owe my thanks to **Dr. A.R. Parveen Gani,** Associate Professor of Community Medicine for her guidance and help during this study.

I am immensely grateful to all the **Patients** who took part in this study.

CONTENTS

INTRODUCTION

INTRODUCTION

Eclampsia (Greek – Shining Forth)

An acute and life threatening complication of pregnancy is characterized by the appearance of tonic clonic seizures, in a patient with pre-eclampsia.[1]

It is estimated to complicate 1in 2000 deliveries in developed countries and from 1 in 100 to 1 in 1,700 deliveries in developing countries.[2]

Eclampsia accounts for 50,000 maternal deaths a year world wide.The maternal case fatality rate is 1.8% and 35% of eclamptics will have one major complication.[3] The perinatal mortality rate in developing countries is as high as 80 (or) more per 1000 births.

Although it is a standard practice to use anticonvulsants for management of Eclampsia, the choice of agent is controversial.[4] Magnesium sulphate was first used in the treatment of puerperal eclampsia in 1925.

Pritchard[5] (1955) published his initial experience with magnesium sulphate in the treatment of eclampsia. The first two randomized trials of anticonvulsant treatment in eclampsia were published in 1990.

The Collaborative Eclampsia Trial (CET) involving 1687 women with eclampsia in the year 1995 provides compelling evidence that magnesium sulphate reduces the risk of recurrent seizures as compared with diazepam and phenytoin and also less maternal and neonatal morbidity than the other agents.[6]

A prospective study included 65 eclamptic patients receiving lower dose of magnesium sulphate therapy at Dhaka Medical College Hospital from 25th March 1998 to 15th June 1998.[7]

The loading dose of Dhaka regimen was significantly less than that used by CET and Pritchard regimen – 10gm loading dose as compared to 14gm and the maintenance dose was 2.5gm intramuscularly 4th hourly which was half of the maintenance dose used in Pritchard regimen. With this regimen, the maternal mortality rate in Dhaka medical college has fallen dramatically.

The present study compares the Pritchard regimen with the Dhaka regimen of magnesium sulphate in the management of Antepartum eclampsia and compares the maternal and perinatal out come in the patients treated with the two regimens.

AIM OF STUDY

AIM OF THE STUDY

Magnesium sulphate has been shown to be the drug of choice for the control of seizures in eclampsia. However, its toxicity, which is linked to serum magnesium level, can be life-threatening for the mother and affects the neonatal outcome. Hence, the aim of the present study is

- To study the safety and efficacy of low-dose magnesium sulphate regimen – Dhaka regimen in the treatment of Antepartum Eclampsia.
- To compare the effects of Dhaka regimen with the Pritchard regimen.
    a) The efficacy of controlling convulsions in Antepartum Eclampsia.
    b) In preventing the recurrence of convulsions.
- To compare the maternal and perinatal outcome with the two regimens – Dhaka regimen and the Pritchard regimen.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Definition

Preeclampsia complicated by generalized tonic-clonic convulsions is termed Eclampsia. Fatal coma without convulsions also is called eclampsia. However, it is better to limit the diagnosis to woman with convulsions and to regard deaths in non convulsions cases as due to severe preeclampsia.[8]

Depending on whether convulsions appear before, during (or) after labour, eclampsia is designated as Antepartum, Intrapartum (or) Post partum eclampsia.

Etiology

The exact etiology of pre eclampsia still remains unknown. Several theories have been proposed. Since eclampsia is a severe form of preeclampsia, the histopathological and biochemical changes are similar although intensified when compared to those of preeclampsia.[9]

According to Sibai[10] (19), currently plausible potential causes include the following,

(i) Abnormal trophoblastic invasion of uterine vessels.

(ii) Immunological intolerance between maternal and fetoplacental tissues.

(iii) Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy.

(iv) Dietary deficiencies

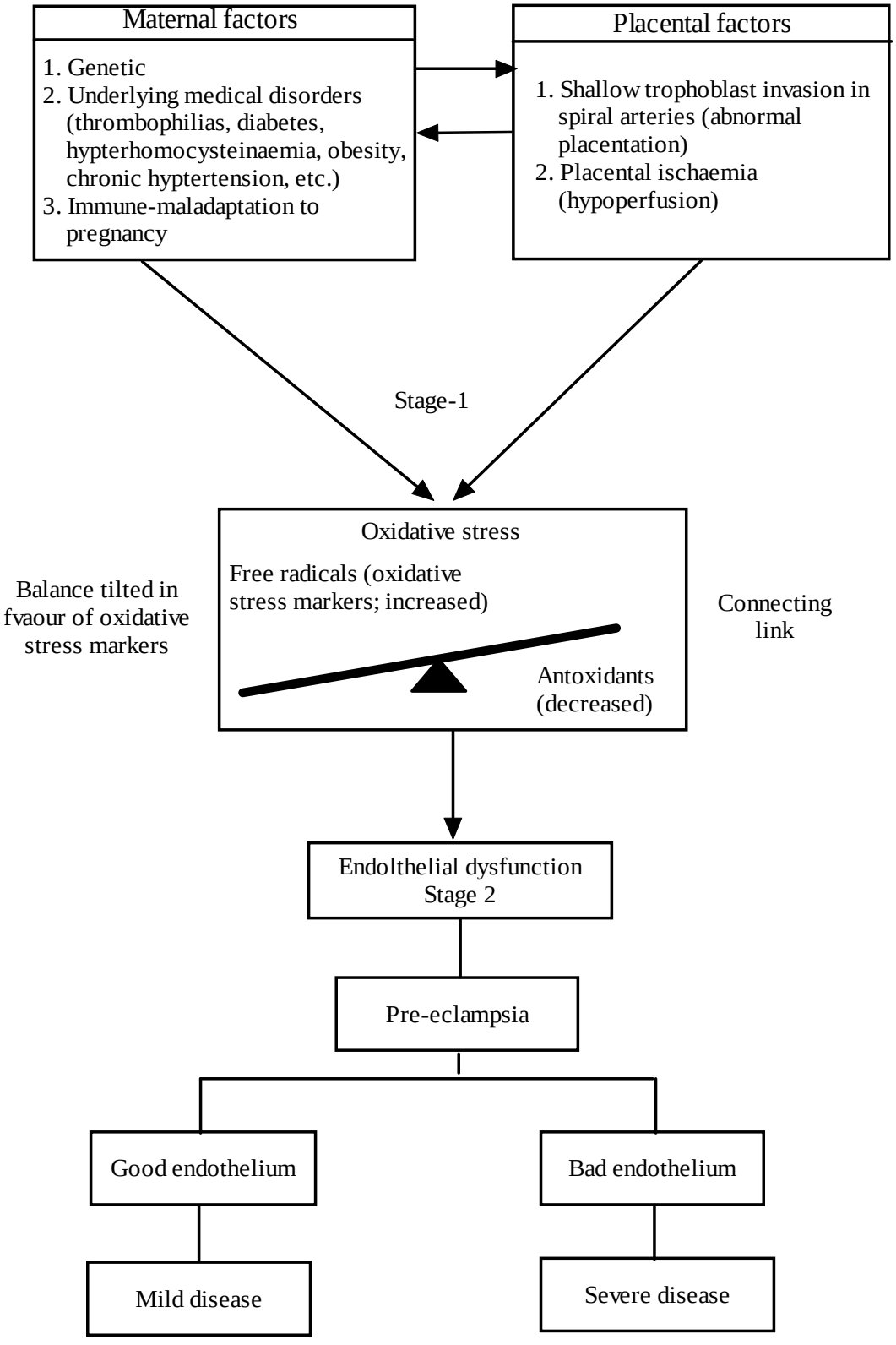
(v) Genetic influences

Table -1 Risk factors for pre-eclampsia[11]

	Relative risk (RR)	**95% Cl**
Genetic factors		
Genetic pre-disposition		
Race and ethnicity: more common in Blacks and Asians.		
Family history of pre-eclampsia	2.90	1.70-4.93
Pregnancy by ovum donation		
Age and parity		
Teenage pregnancy		
Age more than 40 years	1.96	1.34-2.87
Long interval between pregnancies		
Nulliparity	2.91	1.28-6.61
Partner-related factors		
Change of partner		
Partner who fathered a pre-eclamptic pregnancy in another woman		
Limited sperm exposure		
Pregnancy due to donor insemination		
Presence of underlying disorders		
Chronic hypertension		
Diabetes mellitus	3.56	2.54-4.99

Renal disease Obesity (body mass index > 35 kg/m^2) (i) Before pregnancy (ii) At booking Maternal low birth weight Polycystic ovarian syndrome Migraine Collagen vascular disorders Uncontrolled hyperthyroidism Factor V Leiden deficiency, activated protein-C deficiency and thrombophilia Sickle cell disease or trait and other haemoglobinopathies Anti-phospholipid antibodies Protein-S deficiency and hyperhomocysteinaemia	2.47 1.55 9.72	1.66-3.67 1.28-1.88 4.34-21.75
Pregnancy-related risk factors Multiple pregnancy Hydatidiform mole Hydrops fetalis Congenital and chromosomal fetal anomalies (trisomy 13, triploidy) Urinary tract infection	2.93	2.04-4.21
Miscellaneous factors Smoking (reduced risk) Psychological strain and stress at working place Previous history of pre-eclampsia Raised blood pressure (diastolic>80 mmHg) at booking	7.19 1.38	5.85-8.83 1.01-1.87
Cl = Confidence interval		

Genesis of preeclampsia as a two-stage disorder[12]



PATHOGENESIS

1) Vasospasm

The concept of vasospasm was documented by Volhard (1918) based on direct observation of small vessels in nail beds, ocular fundi and bulbar conjunctiva. Wang and colleagues[13] (2002) demonstrated disruption of endothelial junctional proteins. Suzuki *et al.,*[14] (2003) demonstrated the ultra structural changes in sub endothelial region of resistance arteries in preeclamptic woman. With diminished blood flow, ischaemia of the surrounding tissues would lead to necrosis, haemorrhage and other end-organ disturbances as characteristic of the syndrome. Vasospasm may be worse in HELLP syndrome (Fisher *et al*., 2000)[15].

2) Endothelial cell activation

Hayman *et al.*[16] (2000) showed that clinical syndrome of preeclampsia results from widespread endothelial cell changes.

(3) Increased Pressor responses

Abdul – Karim and Assali[17] (1966) showed that normal pregnant women develop refractoriness to infused vasopressors. Women with early preeclampsia have increased vascular reactivity to infused nor epinephrine and angiotensin II. (Raab *et al*., 1956 and Talledo *et al*., 1968).[18]

(4) Prostaglandins

Taylor and Roberts[19] (1999) showed that endothelial prostacyclin production mediated by phospholipase A_2 is decreased in preeclampsia. Thromboxane A_2 secretion by the platelets is increased and the prostacyclin: thromboxane A_2 ratio decreases. This favours increased sensitivity to angiotensin II that ends in vasoconstriction.

Chavarria[20] (2003) gave the evidence that these changes are apparent as early as 22weeks in woman who later develops preeclampsia.

(5) Nitric Oxide

A potent vasodilator synthesized from L-arginine by endothelial cells. It is the compound that maintains the normal low-pressure vasodilated state characteristic of feto placental perfusion – Myat *et al.* [21] (1992).

Wang *et al.* [22] (1992) showed that preeclampsia is associated with decreased endothelial nitric oxide synthase expression, which increases cell permeability. Its production is increased as a compensatory mechanism in severe preeclampsia. So increased serum concentration of nitric oxide is likely the result of hypertension, not the cause (Morris *et al*.1996).

(6) Endothelins

Mastrogiannis and coworkers[23] showed that these 21 amino acid peptides are potent vaso constrictors, and endothelin-1 (ET-I) is the primary isoform produced by human endothelium.

Taylor and Roberts[24] (1999) showed that the placenta is not the source of increased ET-I and it is likely to arise from systemic endothelial activation.

Sagsoz and Kucukozkan[25] (2003) observed that the treatment of preeclamptic woman with magnesium sulphate lowers ET-1 concentrations.

(7) Angiogenic Factors

Vascular Endothelial Growth Factor (VEGF) and Placental Growth Factor (PlGF) are the glycosylated glycoproteins that are selectively mitogenic to endothelial cells. Simmons *et al.* [26] (2000) showed that VEGF is increased in serum from woman with preeclampsia but the bioavailability is decreased. In preeclampsia, the gene for Soluble Fms-Like tyrosine kinase 1 (SFLt) is up regulated – Maynard *et al.* [27](2003)

PATHOPHYSIOLOGY OF ECLAMPSIA

Loss of cerebral vascular autoregulation lead to either overdilatation or intense vasospasm. As part of the autoregulatory response to severe hypertension, cerebral vasoconstriction occurs which leads to ischaemia, cytotoxic edema and infarction. When the autoregulatory mechanism fails at some point, dilatation of vessels occurs resulting in hyper perfusion and vasogenic edema.[28]

Autopsy studies showed edema, cortical and while matter microinfarcts, pericapillary and parenchymal bleeding and vascular lesions predominantly in the occipital and watershed areas.[29]

Symptoms and signs of impending eclampsia

1. Headache - Persistent occipital or frontal
2. Visual disturbance - Blurred vision and photophobia.
3. Restlessness and agitation
4. Epigastric and/or right upper quadrant pain
5. Nausea and vomiting
6. Oliguria
7. Laboratory evidence of disseminated intravascular coagulation.

Clinical Course of eclampsia

Eclamptic seizure as stated by Chesley[30] has 4 stages.

(i) Stage of invasion - The patient becomes unconscious. There is twitching of the muscles of the face, tongue and limbs which lasts for about 30 seconds.

(ii) Stage of contraction - The whole body goes into a tonic spasm. Cyanosis appears. This lasts for about 30 seconds.

(iii) Stage of convulsion - All voluntary muscles undergo alternate contraction and relaxation. Biting of the tongue occurs. This lasts for about 1-4 minutes.

(iv) Stage of coma - Following the convulsions, the patient passes on to the stage of coma which usually lasts for a brief period.

Differential Diagnosis

Epilepsy, Hysteria, Encephalitis, Meningitis, Puerperal cerebral thrombosis, cerebral malaria in tropics, cysticercosis, Intra cranial tumour.

Complications of Eclampsia

Maternal complications

(i) Maternal Injuries

(ii) Placental Abruption (10%)

(iii) Neurological deficits (7%)

(iv) Aspiration pneumonia (7%)

(v) Pulmonary edema (5%)

(vi) Disseminated intra vascular coagulation (3%)

(vii) Cardio pulmonary arrest (4%)

(viii) HELLP syndrome (4%)

(ix) Acute Renal failure (4%)

(x) Maternal death (1%)

Fetal complications

The perinatal morbidity and mortality rate is very high to the extent of about 30 – 50%. The causes are:

(i) IUGR due to chronic placental insufficiency.

(ii) Prematurity – either spontaneous or induced.

(iii) Intra uterine asphyxia

(iv) Effects of the drugs used to control convulsions.

(v) Increased operative deliveries.

MANAGEMENT

I. General Management

It plays an important role in the management of eclampsia. The patient is nursed in a quiet room with a medical or nursing attendant always present. Pulse rate, respiration, blood pressure, oxygen saturation, restlessness, urine output must be constantly observed. A mouth gag, airway and O_2 must be available. Patient is put in left lateral position in a railed cot. Throat is cleared of secretions and vomitus by intermittent suctioning. A soft firm mouth gag introduced in time will save injury to the tongue. An indwelling catheter in the bladder will give an accurate assessment of the urine output and will also prevent restlessness due to a full bladder. Blood pressure is measured half hourly till it is controlled and then second hourly. A record of grade of consciousness is maintained. Nutrition and hydration are maintained parenterally.

II. ANTICONVULSANT LINE OF MANAGEMENT

Dosage schedule of various regimens

1. Menon's Regimen (1961) [31]

Chlorpromazine 25 mg and pethidine 100 mg in 20 ml of 5% glucose is given intravenously along with 50 mg of chlorpromazine and 25 mg promethazine given intramuscularly.

Subsequently, promethazine 25 mg and chlorpromazine 50 mg are given intramuscularly alternatively at four hourly intervals for 24 hrs following the last fit.

Menon (1961) used lytic cocktail in 1448 eclamptic women and maternal mortality rate was 2.2%.

2. Diazepam [32]

A loading dose of 10 mg diazepam intravenously over 2 minutes is followed by an intravenous infusion of 40 mg in 500 ml of normal saline for 24 hours. Rate of infusion titrated against the level of consciousness with the aim of keeping the woman sedated but arousable. Diazepam can cause respiratory depression. It is poorly excreted by the neonates and they tend to be sedated, hypothermic and unable to breast feed for several days. Maternal mortality rate using this regimen was 5%.

3. Magnesium Sulphate ($MgSO_4$)

In 1955, Pritchard initiated a standardized treatment regimen at Parkland Hospital.

In 1964, Zuspan initiated the intravenous magnesium sulphate regimen.

a) Pritchard Regimen [33]

Loading Dose	Maintenance Dose
4 gm of 20% $MgSO_4$ IV at a rate not exceeding 1 gm/min	Every 4 hrs thereafter, 5gm of 50% $MgSO_4$ as IM on alternate buttocks after ensuring
10 gm of 50% $MgSO_4$ as deep IM, 5 gm in each buttock through a 3 inch long – 20 gauge needle	a) Patellar reflex is present b) Respiration rate > 16/minute c) Urine output > 100 ml in the preceding 4 hours
If convulsions persists after 15 minutes, 2 gm of 20% $MgSO_4$ IV is given at a rate not exceeding 1 gm/minute	$MgSO_4$ is continued for 24 hours after delivery or the last episode of convulsion whichever is later.

b) Zuspan's Regimen (1964)

Loading Dose	Maintenance Dose
4 gm of 20% $MgSO_4$ IV at a rate not exceeding 1 gm /min	1 – 2 gm/hour by controlled infusion pump for 24 hours after delivery (concentration not to exceed 20%)

c) Dhaka Regimen[7] (1998)

Loading Dose	Maintenance Dose
4 gm of 20% $MgSO_4$ IV at a rate not exceeding 1 gm/min	Every 4 hrs thereafter, 2.5gm of 50% $MgSO_4$ as IM on alternate buttocks after ensuring
6 gm of 50% $MgSO_4$ as deep IM, 3 gm in each buttock through a 3 inch long – 20 gauge needle	a) Patellar reflex is present b) Respiration rate > 16/minute c) Urine output > 100 ml in the preceding 4 hours

Phenytoin Regimen

It is given by slow intravenous route. Initial doses 10mg / kg body weight followed by 5mg / kg 2hours later. Thereafter 200mg of phenytoin is given orally after 12 hours. It is continued until 48 hours after delivery. Important side effects are hypotension, cardiac arrythmias and phlebitis at the administration site. Experience with phenytoin is limited.

Pharmacokinetics of Magnesium Sulphate [34]

Magnesium sulphate USP is $MgSO_4.7H_2O$. It has a molecular weight of 24.3. 1gm of magnesium sulphate has 98mg of elemental magnesium.

Distribution and plasma levels

After administration, about 40% of plasma magnesium is protein bound. The unbound magnesium ion diffuses into the extravascular – extracellular space, into bone and across the placenta and fetal membrane and into the fetus and amniotic fluid. In pregnant women, apparent volumes of distribution usually reach constant values between the third and fourth hour after administration. The pharmacokinetic profile of $MgSO_4$ after intravenous administration can be described by a 2-compartment model with a rapid distribution (a) phase followed by a relative slow beta phase of elimination.

Excretion

Magnesium is excreted almost solely by the kidneys. 50% of the infused dose is excreted after 4 hours in urine. 90% of the bolus intravenous dose is excreted within 24 hours.

Mechanism of action

Some believe its action to be mainly peripheral at the neuro muscular junction with minimal central effects. While some believe that the main action is central. Calcium entry into the neurons is regulated by specific excitatory amino acid linked channels. Excitatory amino acids such as L-glutamate and L-aspartate are the major neuro transmitters in mammalian central nervous system. These neurotransmitters produce their effects by interacting with certain receptors on the cell surface. The excitatory amino acid receptor, N-methyl D-aspartate (NMDA) is the best characterized excitatory amino acid receptor sub type. NMDA receptor has its channel blocked by magnesium ion and thus blocking neuronal calcium influx[35]. Thus magnesium has a central nervous system effect in blocking the seizure. Cotton *et al.*[36] (1992) have shown that hippocampal seizures could be blocked by magnesium.

Magnesium sulphate is a potent vasodilator especially in cerebral vasculature thus relieving cerebral vasospasm which is thought to be a cause for eclampsia.

Other actions

- Vasodilatation in vascular beds
- Increased uterine blood flow
- Increased renal blood flow
- Increased prostacyclin release by endothelial cells
- Decreased plasma renin activity
- Decreased angiotensin converting enzyme levels
- Attenuation of vascular response to pressor substances
- Bronchodilatation
- Reduced platelet aggregation

Pharmacological effects

- Anti convulsant action
- Transient hypotensive effect
- Transient but mild decrease in frequency of uterine contractions but no change in the intensity of contractions

- Ø Clinically insignificant decrease in short term variability of fetal heart rate
- Ø No change in long term variability of fetal heart rate or fetal heart rate accelerations

Side effects

First sign of magnesium toxicity is usually the loss of patellar reflex that occurs usually at about 9-12mg/dl because of curariform action. So maintenance dose of $MgSO_4$ is not to be given in the absence of patellar reflex.

Early signs and symptoms of magnesium toxicity include nausea, feeling of warmth, flushing, somnolence, double vision, slurred speech and weakness. These symptoms usually develop at plasma levels of 9 to 12 mg/dl. Muscle paralysis and respiratory arrest develop at plasma level of 15-17mg/dl. Hence respiratory rate is monitored closely.

Cardiac arrest develops at a level of 30-35 mg/dl. Thus, it is important to keep an ampoule containing 1 gram of calcium gluconate at the bedside for intravenous administration as an antidote in case of magnesium toxicity.

Endo - tracheal intubation and mechanical ventilation is done for severe respiratory depression (or) arrest. There is a transient decrease in

uterine activity during intravenous injection alone. It can cause a transient decrease in fetal heart rate variability, neonatal neuro muscular and respiratory depression, hyporeflexia and low APGAR scores. These effects were reported in preterm infants in association with fetal growth restriction, but no ill effects in term infants.

Efficacy and safety

Magnesium sulphate is rapidly effective, reliable and with predictable duration of action, wide safety margin, non depressive and non toxic to the mother and baby, simple to administer and monitor in the clinical setting and with a readily available antidote. Serum magnesium can be measured to ensure therapeutic concentration but many practitioners are happy to omit biochemical monitoring because of its wide margin of safety.

Duley *et al*.[37] (1995) in his study used clinical evaluation alone and showed that the there is no need to check serum magnesium levels. Estimation of magnesium levels is useful in the management of treatment failures.

Drug interactions with Magnesium Sulphate

Agent	Effect	Recommendation
Depolarising/non depolarizing neuro muscular blockers.	Increased activity of these agents.	May need dosage reduction of neuro muscular blocking agents.
CNS depressants - opioids, barbiturates, general anaesthetics	Additive CNS depression	May require dose reduction of CNS depressants
Nifedipine	Hypotension	Administer with caution and adjust nifedipine dosage if necessary

At the neuromuscular junction, magnesium decreases the presynaptic release of acetylcholine and reduces the sensitivity of the post junctional membrane (motor end plate). Ghoneim and Long reported that the actions of succinylcholine (non depolarizing agent) are potentiated by magnesium sulphate. A single dose of succinylcholine can be safely used to facilitate tracheal intubation but may not apply when repeated doses of succinyl choline are used.

When a patient is simultaneously exposed to magnesium sulphate and nifedipine, some interaction might be expected as both are calcium channel blockers. Women on nifedipine who are receiving magnesium

sulphate had effective blood pressure control without undesirable side effects and no cases of hypotension. It would appear that while a theoretical risk of interaction could exist, in practice this is relatively uncommon.

Anti-hypertensive agents used in pregnancy [38]

Class	Drug	Initiating dose	Max dose	Common side effects
Centrally Acting	Methyldopa	250mg tds	2g	Postural hypotension, drowsiness, dryness of mouth, headache, depression.
Calcium channel blockers	Nifedipine	10mg tds	120mg	Headache, dizziness, fatigue, flushing, palpitations heart burn, constipation, peripheral edema.
β adrenergic blockers	Labetolol (α & β) Atenolol	100mg bd 50mg od	2400mg 200mg	dizziness, drowsiness, fatigue, bradycardia depression
Vasodilator	Hydralazine	25mg tds	300mg	Flushing, headache, vomiting, diarrhoea

III. OBSTETRIC MANAGEMENT[39, 40]

The definitive treatment of eclampsia is delivery. Attempts to prolong pregnancy in order to improve fetal maturity are unlikely to be of value. Once seizures are controlled, severe hypertension treated, and hypoxia corrected, delivery can be expedited. Vaginal delivery should be considered but cesarean section is likely to be required in primigravidae remote from term with an unfavourable cervix. Vaginal prostaglandins increase the success of induction and augmentation of labour. Hypertension monitoring and control should be continued vigilantly throughout labour.

Cesarean section is done for the following indications:

1. All deeply unconscious patients (unless delivery is imminent)
2. All un co-operative patients due to restlessness.
3. If vaginal delivery is unlikely to occur with in 6-8 hr of the onset of first eclamptic seizure.
4. There is an obstetric indication for a cesarean section.
5. Fetal distress.

Principles of Vaginal Delivery [42]

Second stage of labour should be short and elective operative vaginal delivery can be considered. The third stage of labour should be

managed by either of the following so as to prevent post partum hemorrhage.

i). Oxytocin 10 units in iv drip or im

ii). Prostaglandin F2α 125μg or 250μg im

iii). Misoprostol 600 - 800μg per rectal.

Methyl ergometrine is contraindicated as this would result in further increase in blood pressure.

Post Delivery

After delivery, close monitoring should be continued for a minimum of 24 – 48 hours. Since almost 20% of the patients can have post partum eclampsia – it is important to be vigilant and continue treatment for first 24 – 48 hours. Anti hypertensive treatment can then be gradually tapered off.

MATERIALS AND METHODS

MATERIALS & METHODS

This study was conducted in Annal Gandhi memorial Government Hospital, Tiruchirappalli in the department of Obstetrics and Gynaecology during the period of July 2008 – August 2009.

80 consecutive patients with antepartum eclampsia were included in the study. Magnesium sulphate was used for the control of convulsions. 40 patients were put under the Pritchard regimen and other 40 were enrolled under Dhaka regimen.

Inclusion criteria

All patients with antepartum eclampsia irrespective of their age, parity and booking status.

Exclusion criteria

1. Patients having received $MgSO_4$ before coming to hospital.
2. Patients with preexisting seizure disorder, heart block, renal failure.
3. Postpartum eclampsia with onset of convulsions >72 hrs after delivery.

Design of the study

Randomized control trial with randomization done using list given by statistician.

Group A

40 cases of Antepartum eclampsia were randomly allocated to Dhaka Regimen.

Group B

40 cases of Antepartum eclampsia were randomly allocated to Pritchard Regimen.

History

A detailed history regarding age, parity, gestational age, number of convulsions, duration of symptoms of pregnancy induced Hypertension, H/0 imminent symptoms were taken from close relations and also from the patient if she is conscious (or) taken retrospectively from her. Any past history of hypertension (or) renal disease (or) eclampsia in previous pregnancy was elicited.

Clinical Examination

A thorough general examination and obstetric examination were made. On general examination, conscious level, degree of edema, anaemia, pulse rate, temperature, respiratory rate, blood pressure, cardiovascular system, respiratory system, fundus examination were done. Blood and urine were sent for all investigations related to eclampsia like renal function tests, liver function tests, hematological tests and coagulation screening tests were carried out in all patients.

A life line was established and the Regimen was started. Pulse, Blood pressure, Respiratory rate, Oxygen saturation monitored for every 15 minutes, Knee jerk and urine output every half hourly. Serum magnesium levels measured 3 to 4 hours after the loading dose.

ANTI CONVULSANT LINE OF MANAGEMENT

1) DHAKA REGIMEN OF MAGNESIUM SULPHATE REGIMEN:

Loading Dose

- 4gm of 20% magnesium sulphate given intravenously slowly over 15 minutes.
- 3gm of 50% magnesium sulphate given intramuscularly in each buttock.

Maintenance Dose

- 2.5gm of 50% magnesium sulphate given intramuscularly every 4 hours in alternate buttocks, until 24 hrs after delivery or last episode of fits whichever is later, provided patellar reflex is present, respiratory rate is more than 16/min, urine output at least 100ml in the preceding 4 hours.

2) PRITCHARD'S MAGNESIUM SULPHATE REGIMEN

4gm of Magnesium sulphate ($MgSO_4.\ 7H_2O$, USP) as a 20% solution intravenously at a rate not to exceed 1gm/min, followed promptly with 10gm of 50% magnesium sulphate solution, 5gm deep IM in each buttock.

5gm of 50% solution of magnesium sulphate intramuscularly in alternate buttock every 4 hours thereafter for 24 hours after delivery or last fits whichever is later, provided

a) Patellar reflex is present.

b) Respiratory rate > 16/min

c) Urine output in the previous 4hours exceeded 100ml.

Anti Hypertensive Line of Management

Control of Hypertension is achieved by Tab. Alphamethyl Dopa 250mg / 500mg thrice daily and Tab. Nifidipine 10mg thrice daily.

Obstetric Management

After stabilizing the patient, a detailed obstetric examination was done. Mode of termination was planned according to the gestational age, viability of the fetus, and the cervical scoring.

Patients were induced with prostaglandin E_2 gel and accelerated with Oxytocin infusion.

Cesarean section was done for obstetric indications (or) for failed induction.

After delivery the patient was observed carefully for 48 – 72 hours in the labour ward and post operative ward and followed up until the discharge of the patient.

Neonatal outcome was recorded in terms of Apgar scoring and birth weight. Neonates were also followed up until the discharge of the mother.

Outcome measures

Primary outcome measures are recurrence of fits after starting the treatment in both the regimens. Perinatal morbidity and mortality and maternal morbidity and mortality were compared in both the groups.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

TABLE – 1

AGE DISTRIBUTION

S. No.	Age Group	Group A (Dhaka regimen)		Group B (Pritchard regimen)	
		No.	%	No.	%
1.	Below 20	06	15	01	2.5
2.	20 – 24	18	45	19	47.50
3.	25 – 29	15	37.50	13	32.50
4.	30 & Above	01	2.50	07	17.50
	Total	40	100	40	100
	Mean	24.1625		24.3780	

Age of women in the two groups does not differ significantly. In this study, 7 patients were below 20 years, 37 patients were between 20-24 years, 28 patients were between 25-29 years and 8 patients were 30 years and above. The mean age for Dhaka regimen group was 24.2 years and mean age for Pritchard regimen group was 24.4 years which does not differ significantly.

FIG. 1

AGE DISTRIBUTION

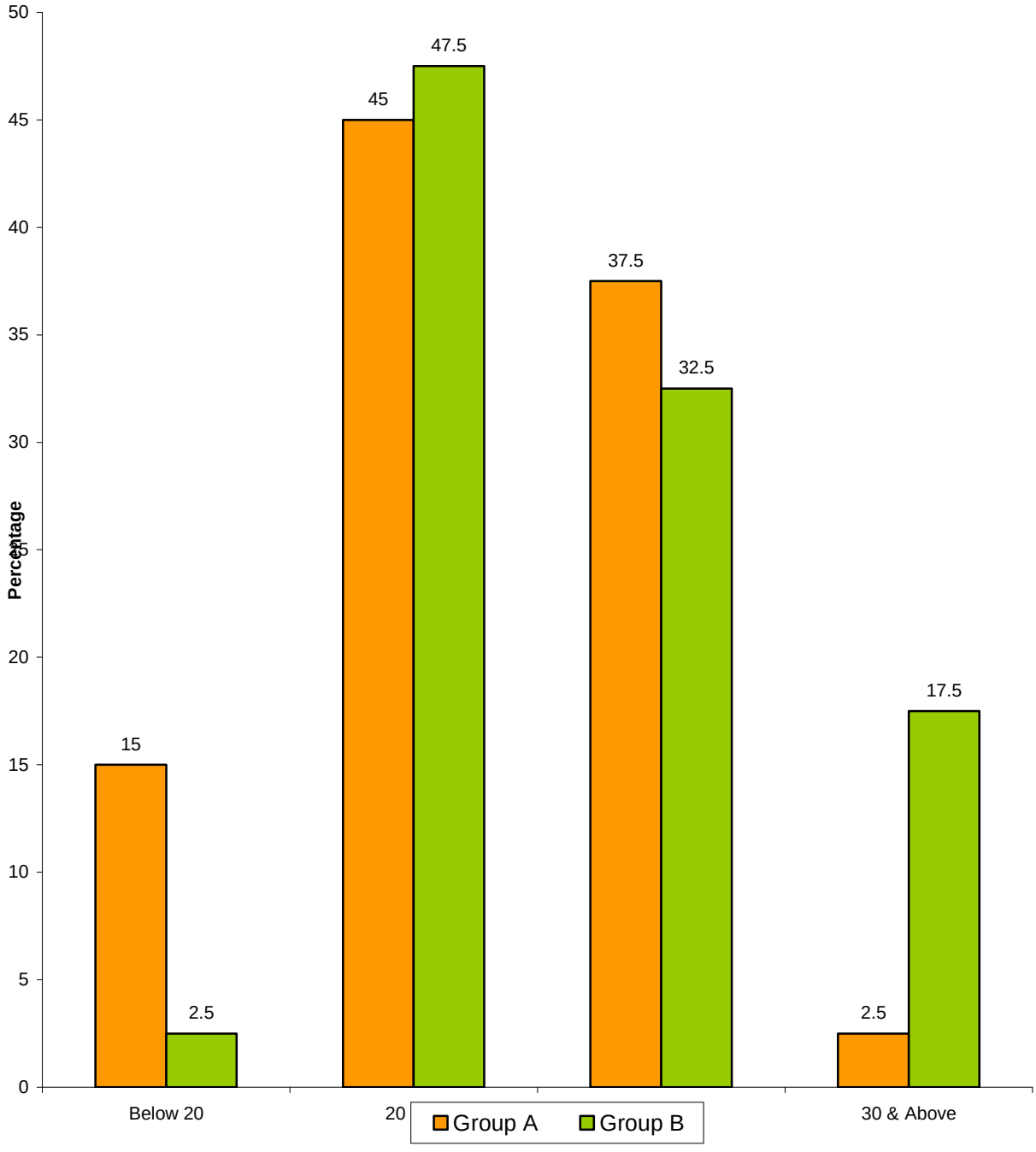


TABLE – 2

BOOKING STATUS

S. No.	Booking Status	Group A (Dhaka regimen)		Group B (Pritchard regimen)	
		No.	%	No.	%
1.	Booked	28	70	27	65
2.	Unbooked	12	30	13	35

In the Dhaka regimen group, 28 patients were booked and 12 patients were unbooked and in the Pritchard regimen group, 27 patients were booked and 13 patients were unbooked. The booking status does not differ significantly between the two groups.

FIG. 2

BOOKING STATUS

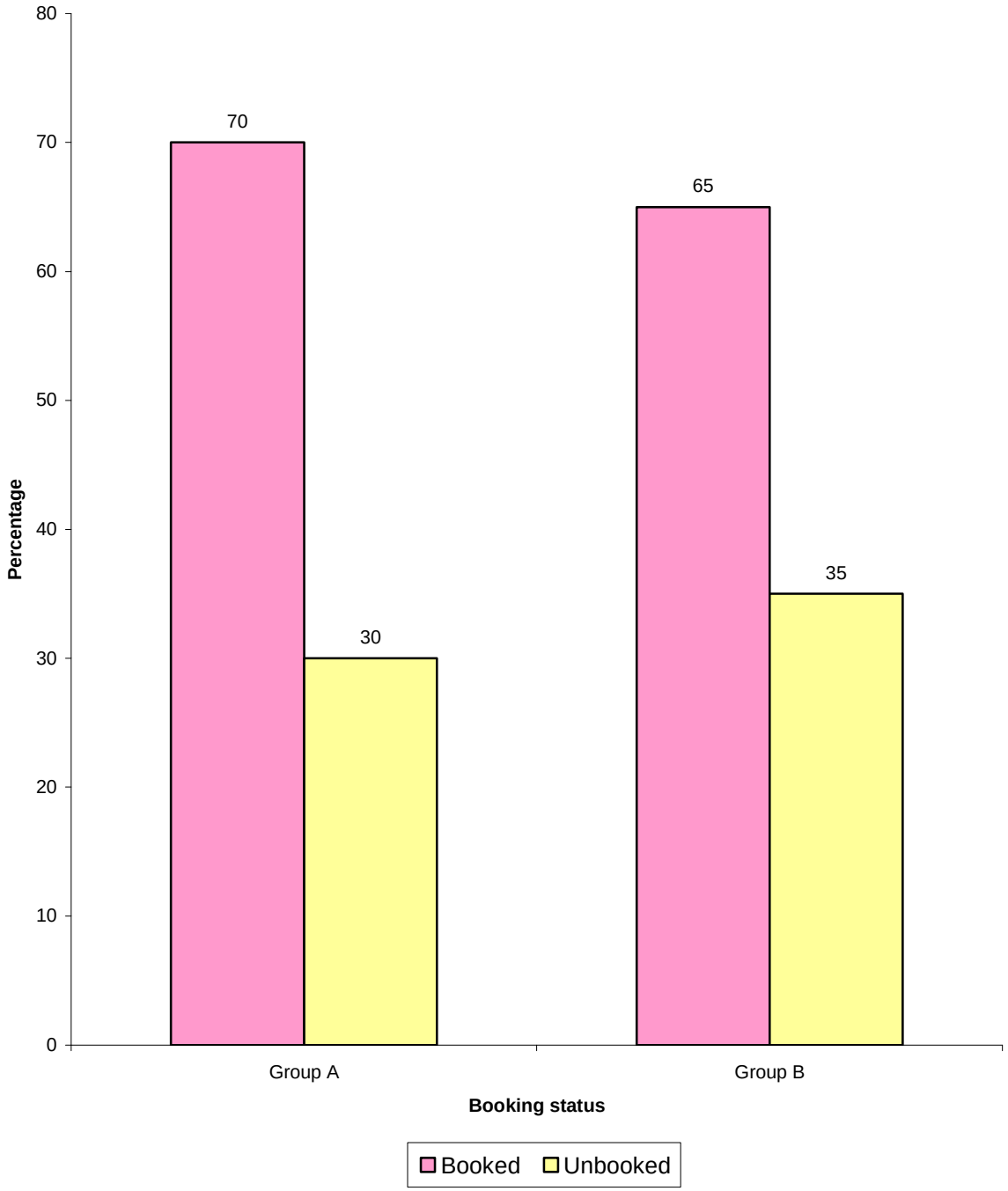


TABLE – 3

PARITY

S. No.	Parity	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
		No.	%	No.	%
1.	Primi	27	67.50	24	60.00
2.	Multi	13	32.50	16	40.00

In this study, 51 patients were pimigravidae. In Dhaka regimen group, 27 patients (67.5%) were primigravidae and 13 patients were multigravidae and in the Pritchard regimen group, 24 patients (60%) were primigravidae and 16 patients were multigravidae. Hence, the parity between the two groups does not differ significantly.

FIG. 3

PARITY

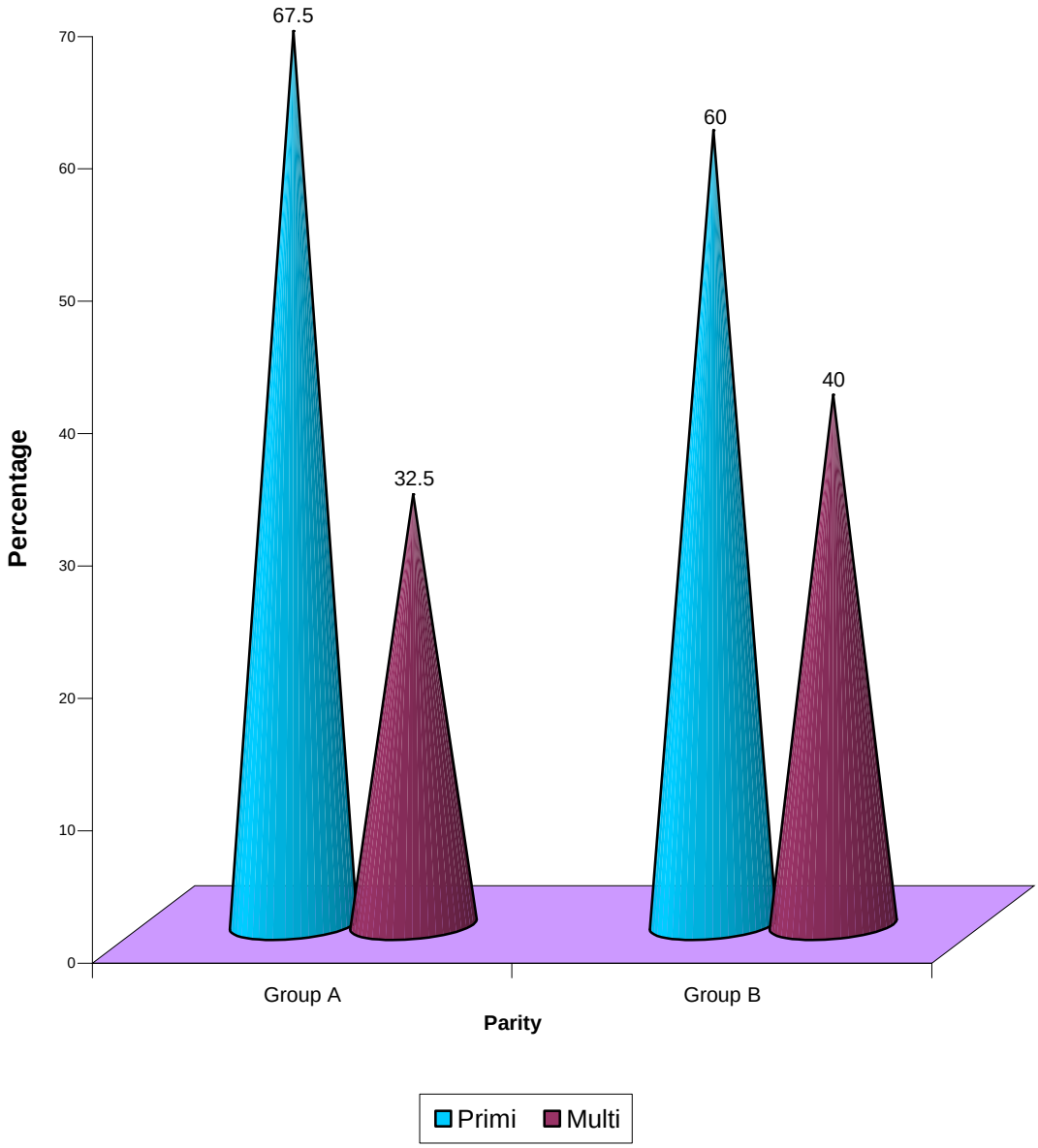

67.5
60
40
32.5
70
60
50
40
30
20
10
0
Percentage
Group A
Group B
Parity
Primi
Multi

TABLE – 4

GESTATIONAL AGE

S. No.	Gestational Age	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
		No.	%	No.	%
1.	Below 24 wks.	04	10.00	00	0
2.	25 – 28 wks.	06	15.00	02	5.0
3.	29 – 32 wks.	09	22.50	14	35.00
4.	33 – 36 wks.	19	47.50	19	47.50
5.	Above 36 wks.	02	5.0	05	12.50
	Mean	32.9375		33.3953	
	P	0.310 (in significant)			

In this study, in Dhaka regimen group, 4 patients were below 24 weeks of gestation, 6 patients were between 25 and 28 weeks, 9 patients were between 29 and 32 weeks, 19 patients were between 33 and 36 weeks, 2 patients were above 36 weeks of gestational age.

In Pritchard regimen group, none of the patients were below 24 weeks, 2 patients were between 25 and 28 weeks, 14 patients were between 29 and 32 weeks, 19 patients were between 33 and 36 weeks, 5 patients were above 36 weeks of gestational age.

The Mean gestational age for Group A (Dhaka Regimen) is 32.94 weeks and Group B (Pritchard regimen) is 33.40 weeks. The P value is 0.310, which is insignificant.

FIG. 4

GESTATIONAL AGE

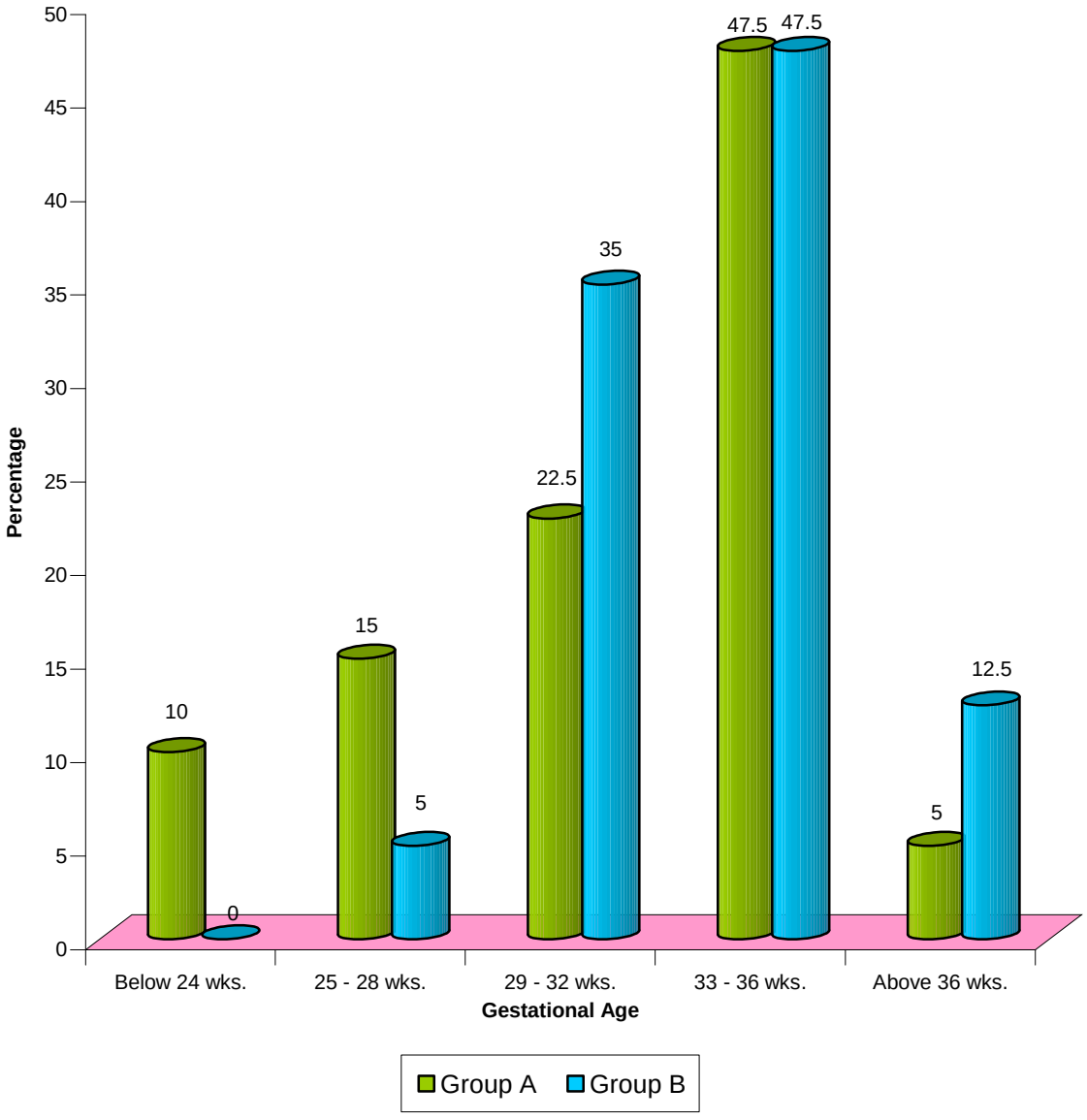


TABLE – 5

NO. OF FITS BEFORE ADMISSION

S. No.	No. of Fits Before Admission	Group A Dhaka Regimen		Group B Pritchard Regimen	
		No.	%	No.	%
1.	1 – 2	18	45.00	12	30.00
2.	3 – 5	17	42.50	25	62.50
3.	6 – 8	05	12.50	03	07.50
4.	Above 9	0	0	0	0

Total number of fits before admission was ranged between 1 and 9 fits. In Dhaka regimen group 18 patients had 1 to 2 fits, 17 patients had 3 to 5 fits, 5 patients had 6 to 8 fits and none had more than that.

In Pritchard regimen group, 12 patients had 1 to 2 fits, 25 patients had 3 to 5 fits, 3 patients had 6 to 8 fits and none had more than that.

FIG. 5

NO. OF FITS BEFORE ADMISSION

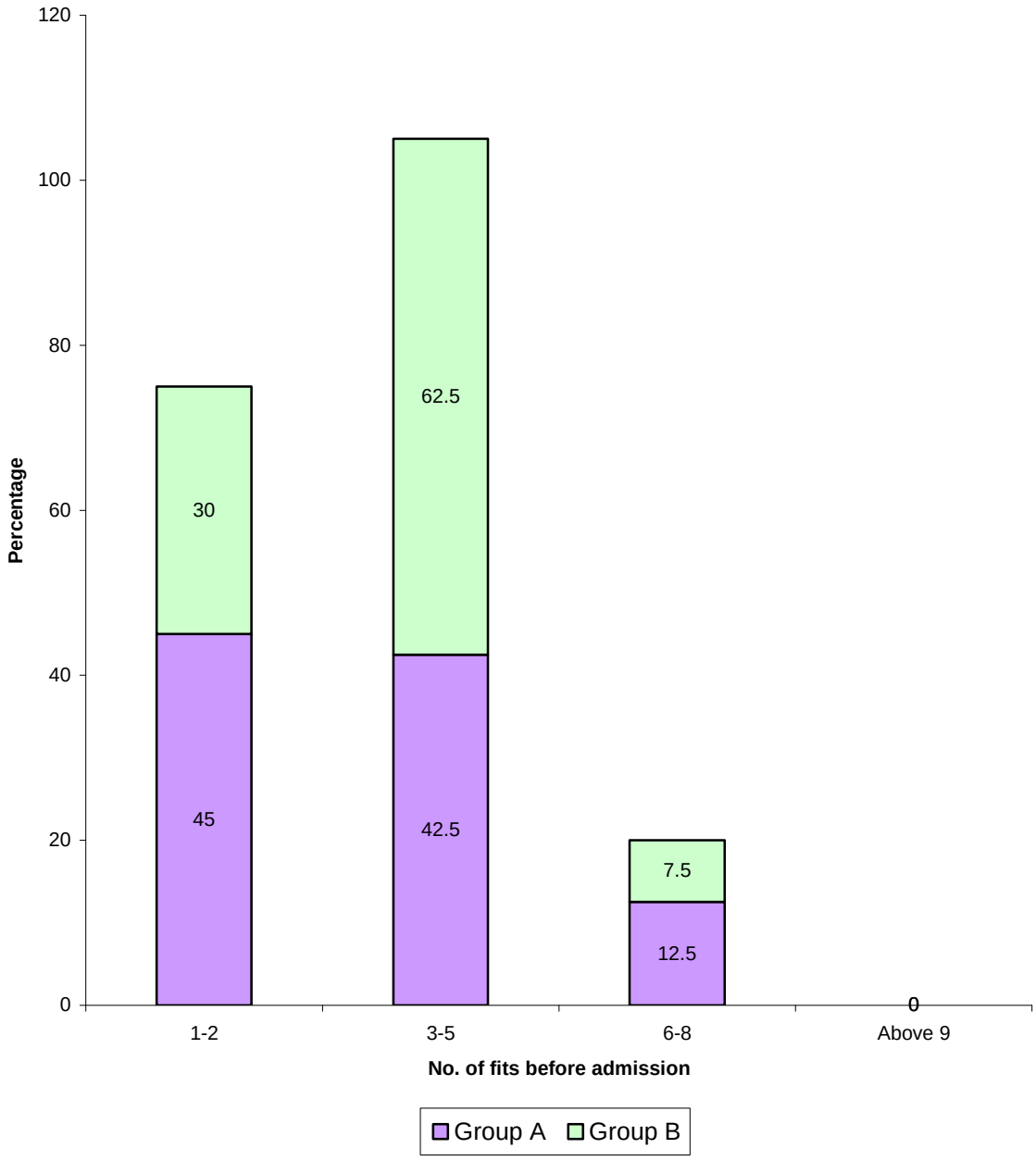


TABLE – 6

LEVEL OF CONSCIOUSNESS

S. No.	Level of Consciousness	Group A Dhaka Regimen		Group B Pritchard Regimen	
		No.	%	No.	%
1.	Conscious	10	25	09	22.50
2.	Semiconscious	30	75	31	77.50
3.	P Value	0.503			

In Dhaka regimen group, 10 patients were conscious and 30 patients were semiconscious. In Pritchard regimen group, 9 patients were conscious and 31 patients were semiconscious. The level of consciousness of the mothers in both the groups does not differ significantly. The P value is 0.503.

FIG. 6

LEVEL OF CONSCIOUSNESS

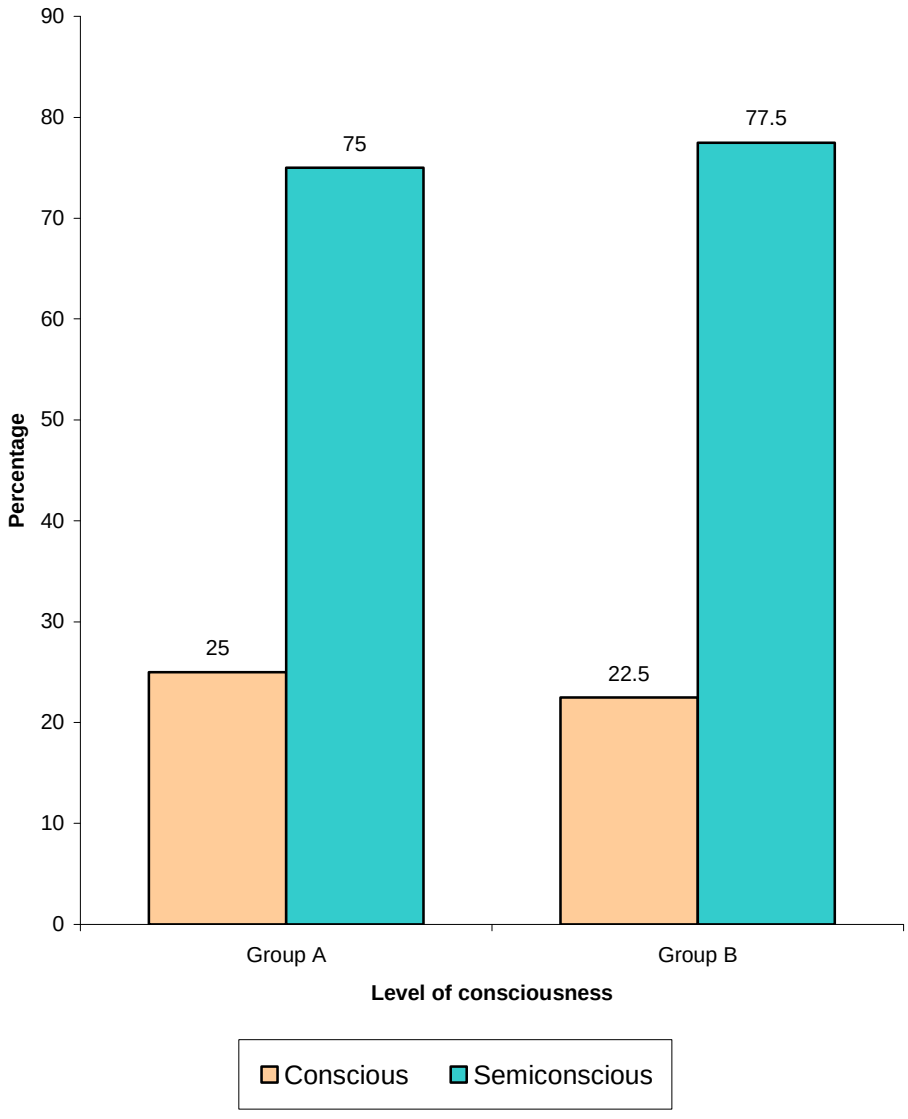


TABLE – 7

HYPERTENSION

S. No.	Blood Pressure (mm Hg)	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
		No.	%	No.	%
	Systolic				
1.	120 – 140	02	5.00	06	15.00
2.	140 – 160	17	42.50	15	37.50
3.	Above 160	21	52.50	19	47.50
	Diastolic				
4.	80 – 100	13	32.50	13	32.50
5.	100 – 110	17	42.50	13	32.50
6.	Above 110	10	25.00	14	35.00

In Dhaka regimen group, two patients had systolic blood pressure less than 140 mm Hg and majority had more than 160 mm Hg. In Pritchard regimen group, 6 patients had systolic blood pressure less than 140 mm Hg and 19 patients had more than 160 mm Hg and the rest had between 140 and 160 mm Hg.

In Dhaka regimen group, 17 patients had diastolic blood pressure between 100 and 110 mm Hg and 10 patients had more than 110mm Hg. In Pritchard regimen group, 13 patients had diastolic blood pressure between 100 and 110 mm Hg and 14 patients had more than 110 mm Hg.

Hence, the blood pressure does not differ significantly between the two groups.

FIG. 7A

SYSTOLIC BLOOD PRESSURE

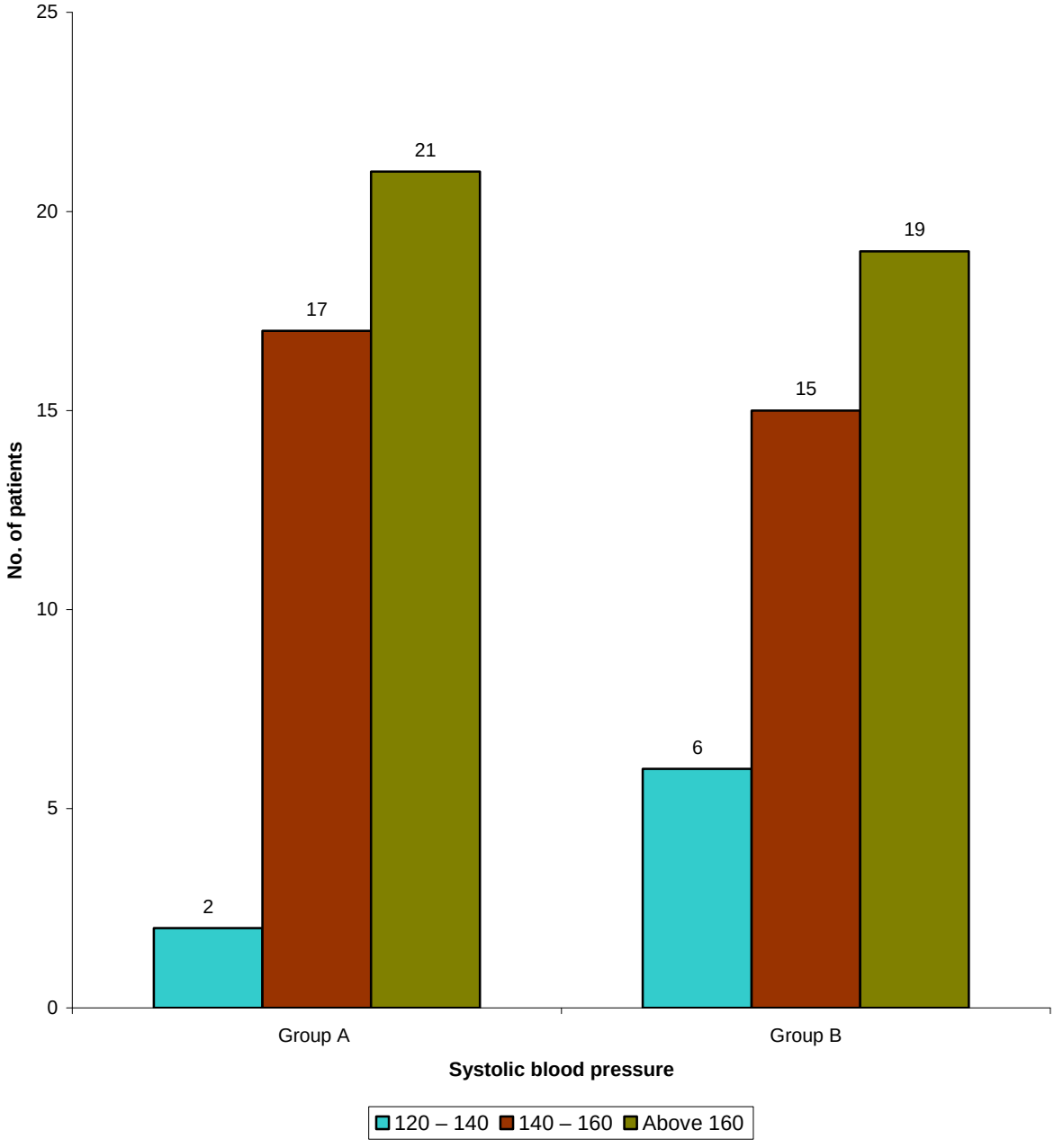


FIG. 7B

DIASTOLIC BLOOD PRESSURE

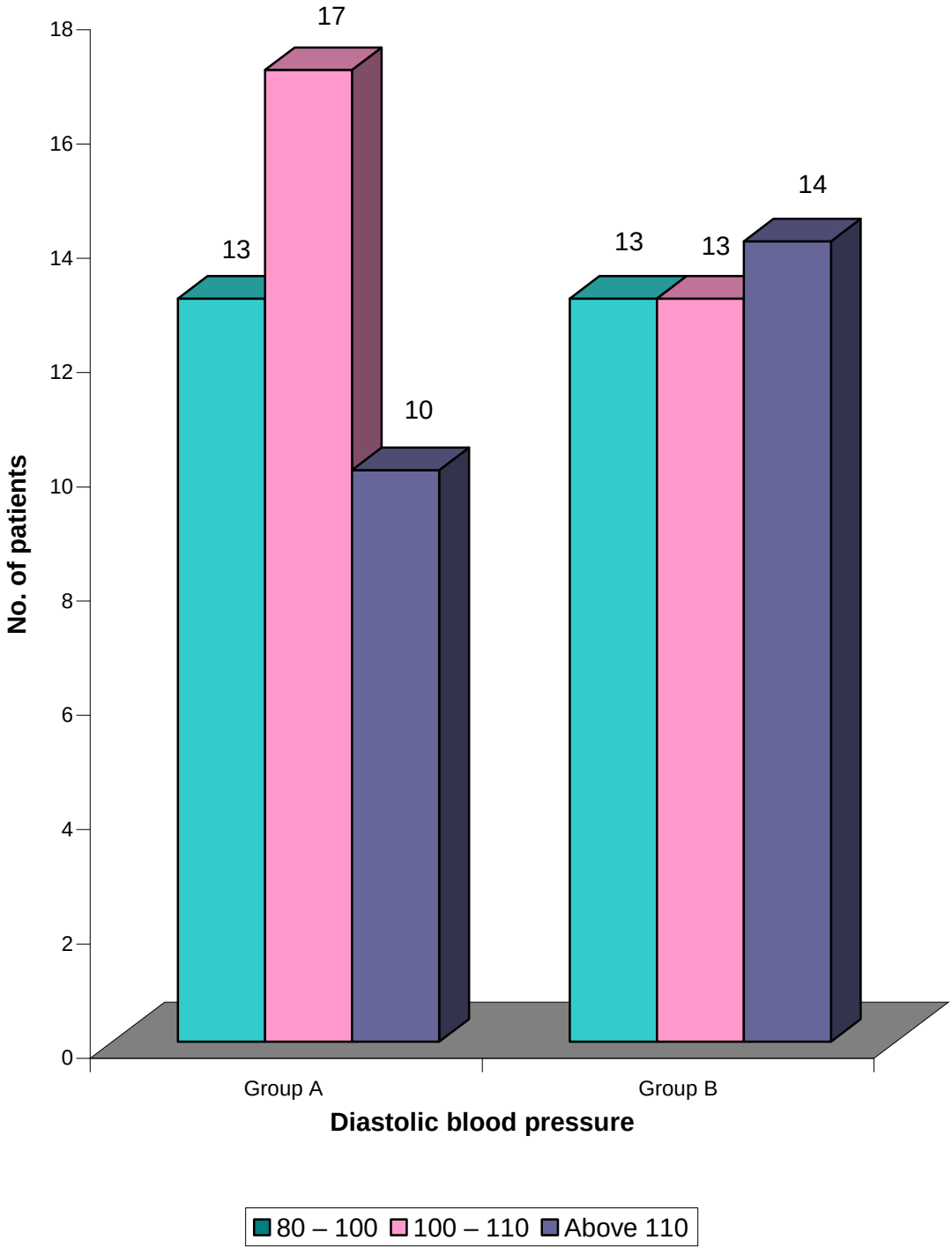


TABLE – 8

SERUM MAGNESIUM MG / DL

S. No.	Serum Magnesium	Group A (Dhaka Regimen)	Group B (Pritchard Regimen)
1.	Mean	4.0338	4.5338
2.	Standard Deviation	0.55250	0.65525
3.	P Value	0.0270	

The mean serum magnesium level in Dhaka regimen group was 4.03 mg/dl and the mean serum magnesium level in Pritchard regimen group was 4.53 mg/dl. Both were within the therapeutic levels without going for toxicity. The P value is 0.0270 which is significant.

TABLE – 9

MODE OF INDUCTION

S. No.	Mode of Induction	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
		No.	%	No.	%
1.	Syntocinon	27	67.50	24	60
2.	Prostaglandin E_2 gel	13	32.50	16	40

In Dhaka regimen group 27 patients were induced with syntocinon and 13 patients were induced with Prostaglandin E_2 gel.

In Pritchard regimen group 24 patients were induced with syntocinon and 16 patients with Prostaglandin E_2 gel.

FIG. 8

MODE OF INDUCTION

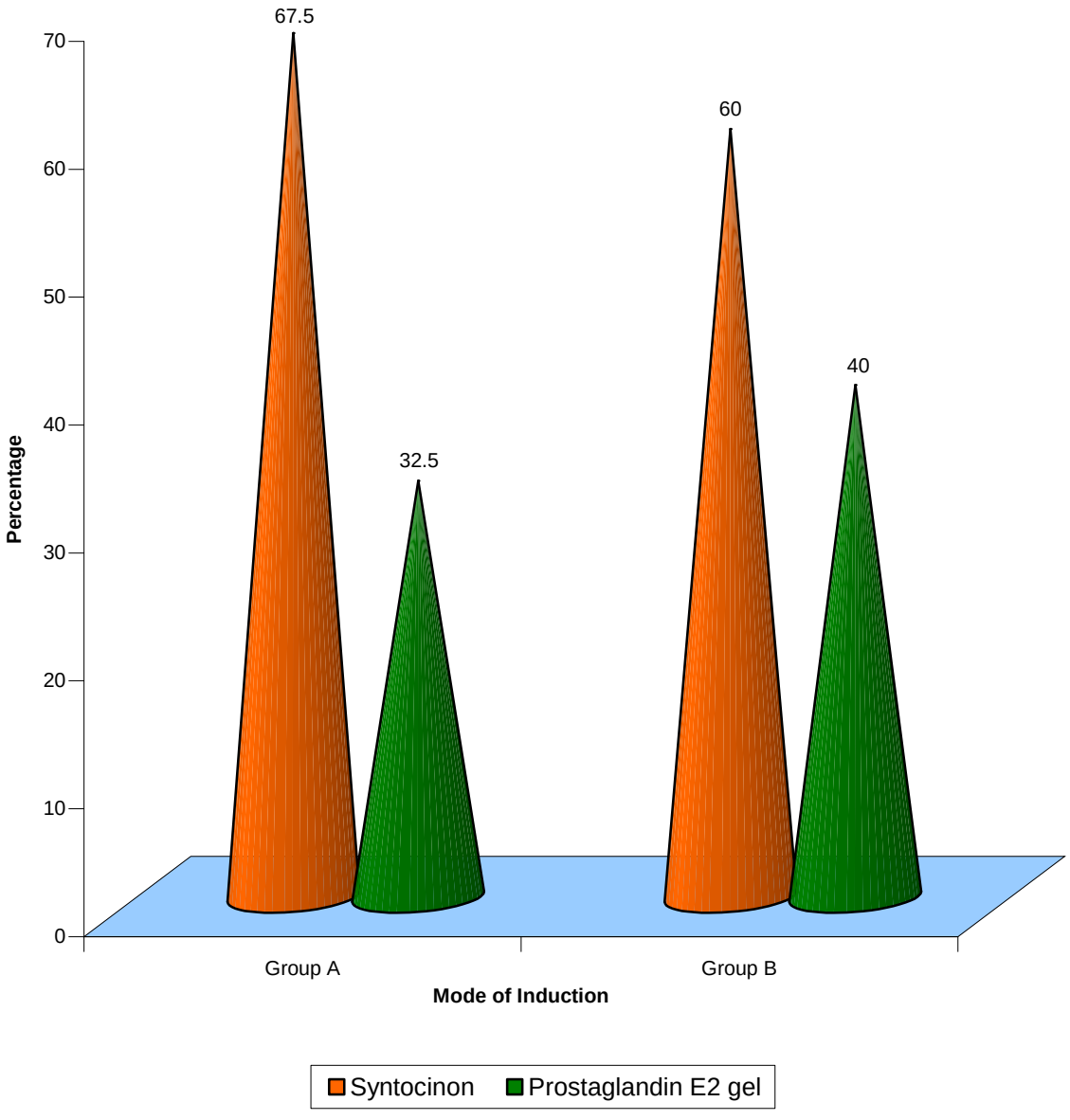


TABLE – 10

MATERNAL OUTCOME IN THE TWO GROUPS

S. No.	Mode of Delivery	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
		No.	**%**	**No.**	**%**
	Vaginal	**26**	**65**	**27**	**67.5**
1.	LN Delivery	22	55	20	50
2.	Outlet forceps	03	7.5	05	12.5
3.	Assisted Breech delivery	01	2.5	02	5.0
	LSCS	**14**	**35**	**13**	**32.5**
	P Value	0.816 (Not Significant)			

Out of 40 patients in Dhaka regimen group 22 patients delivered by Labour Natural, 3 patients by outlet forceps and 1 patient by Assisted Breech delivery and 14 patients delivered by emergency LSCS.

Out of 40 patients in Pritchard regimen group 20 patients delivered by Labour Natural, 5 patients by outlet forceps and 2 patients by Assisted Breech delivery and 13 patients delivered by emergency LSCS.

The P value is 0.816, which is not significant.

FIG. 9

MATERNAL OUTCOME IN THE TWO GROUPS

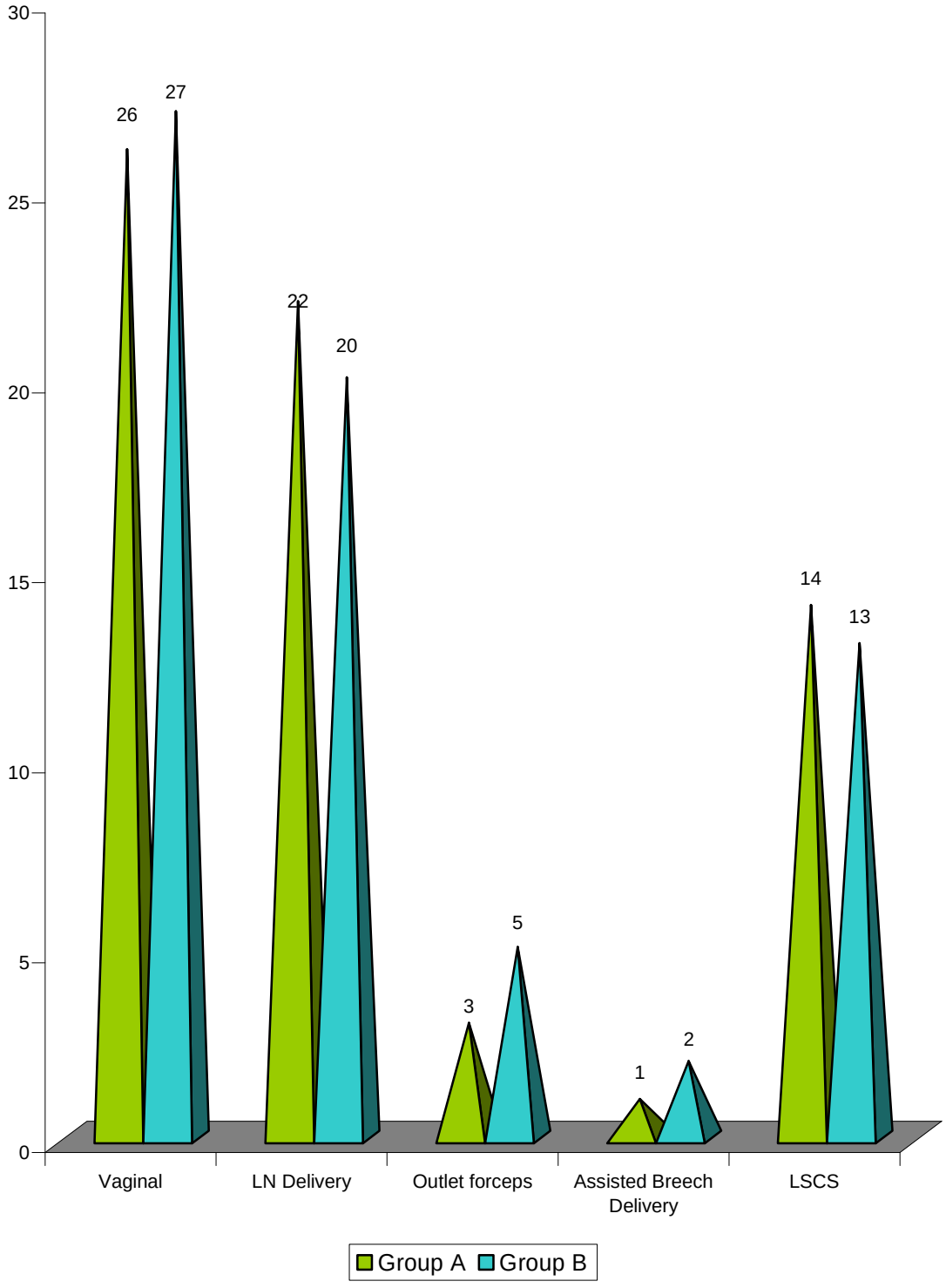


TABLE – 11

INDUCTION TO DELIVERY INTERVAL

S. No.	Induction to Delivery Interval	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
		No.	%	No.	%
1.	Less than 6 hrs.	13	32.50	18	45.00
2.	6.1 – 12 hrs.	27	67.50	21	52.50
3.	12.1 – 18 hrs.	00	00.00	01	02.50
	Mean	12.210		12.816	
4.	Standard Deviation	3.2852		3.821	
5.	P Value	0.123			

The Mean duration of Induction to delivery interval in Dhaka regimen group was 12.21 hours and in Pritchard regimen group it was 12.82 hours. The P value is 0.123 which is not significant.

FIG. 10

INDUCTION TO DELIVERY INTERVAL

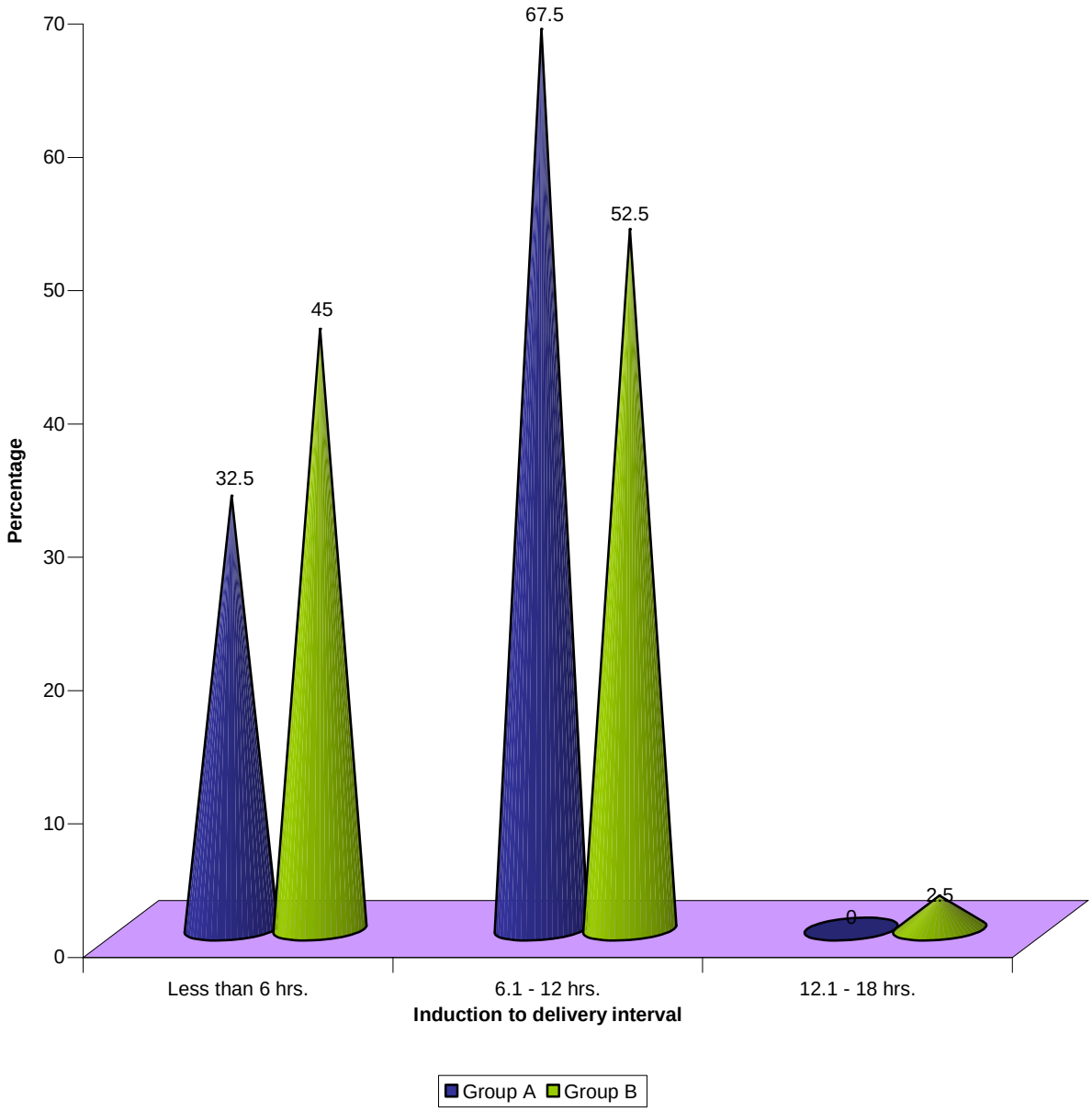


TABLE – 12

TREATMENT COMPLICATIONS IN WOMEN RECEIVING MAGNESIUM SULPHATE FOR ECLAMPSIA

S. No.	Variables	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)		P
		No.	%	No.	%	
1.	Loss of knee jerk reflex	02	5.0	05	12.50	0.02
2.	Oliguria	01	2.5	04	10.00	0.04
3.	Seizure recurrence	02	5.0	01	2.50	0.12
4.	Number of patients required dose deferral	03	7.5	09	2.50	0.03

In Dhaka regimen group, 2 patients had loss of knee jerk reflex, 1 patient had oliguria and 2 patients had seizure recurrence. In Pritchard regimen group, 5 patients had loss of knee jerk reflex, 4 patients had oliguria and 1 patient had seizure recurrence.

In Pritchard regimen group 9 patients required dose deferral which is significantly higher than the Dhaka regimen group, in which only 3 patients required dose deferral due to loss of knee jerk reflex and oliguria.

FIG. 11

TREATMENT COMPLICATIONS IN WOMEN RECEIVING MAGNESIUM SULPHATE FOR ECLAMPSIA

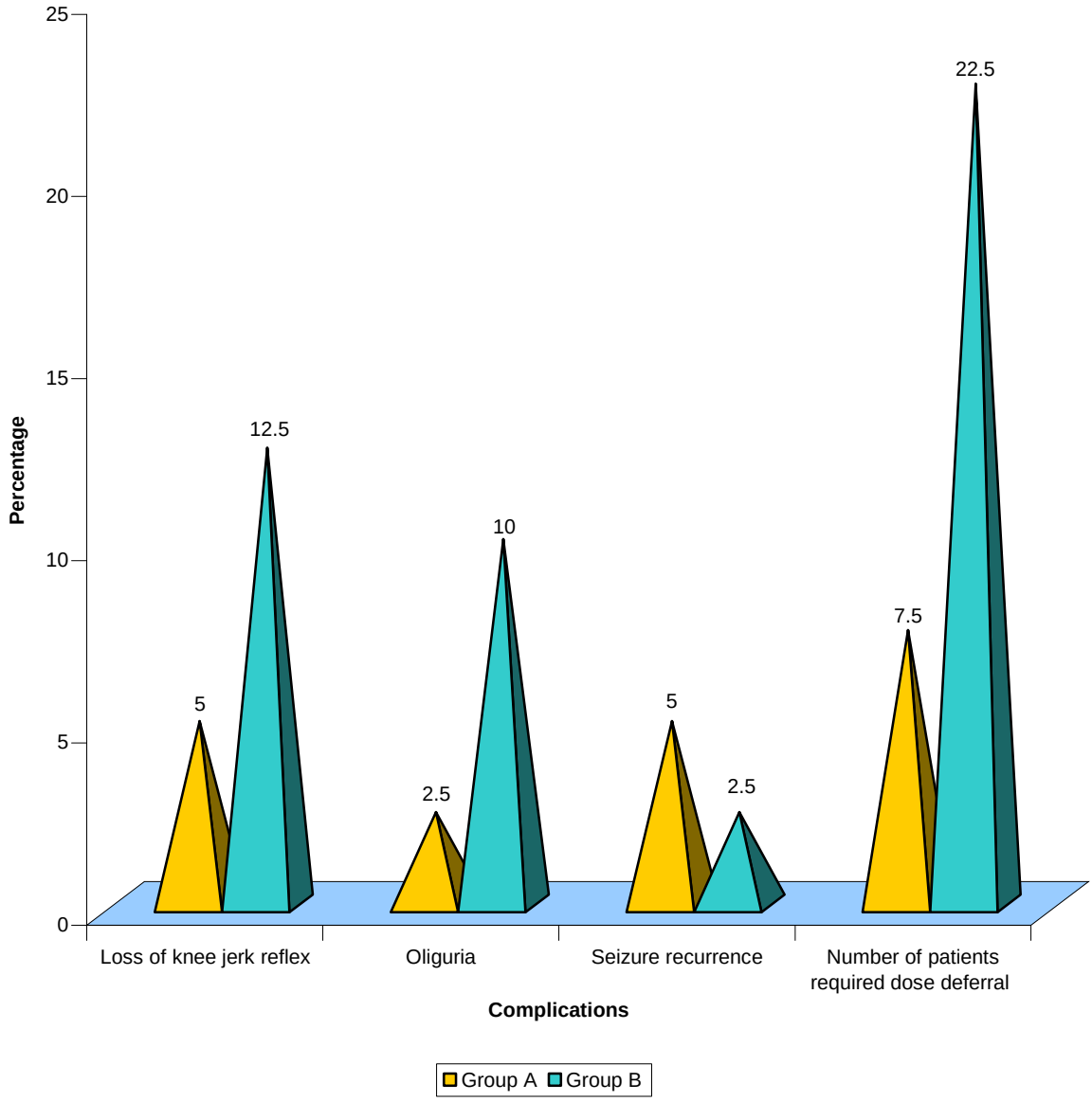


TABLE – 13

MATERNAL CONDITION AT DISCHARGE

S. No.	Condition of the Mother	Group A		Group B	
		No.	%	No.	%
1.	Alive	40	100	40	100
2.	Residual Sequelae	00	00	00	00
3.	Dead	00	00	00	00

In both the treatment groups, all mothers were alive at the time of discharge and none had residual sequlae and there was no maternal mortality in both the regimen groups.

TABLE – 14

PERINATAL OUTCOME

S. No.	Condition of Child after Delivery	Group A (Dhaka regimen)		Group B (Pritchard regimen)	
		No.	%	No.	%
1.	Alive	18	45.00	20	50.00
2.	Still Born	13	32.50	09	22.50
3.	Neonatal death	09	22.50	11	27.50
4.	P Value	0.114 (Not Significant)			

In the Dhaka regimen group, 18 babies were alive, 13 babies were still born and 9 neonatal deaths. In the Pritchard regimen group 20 babies were alive, 9 babies were still born and 11 neonatal deaths. The P value is 0.114 which is not significant.

FIG. 12

PERINATAL OUTCOME

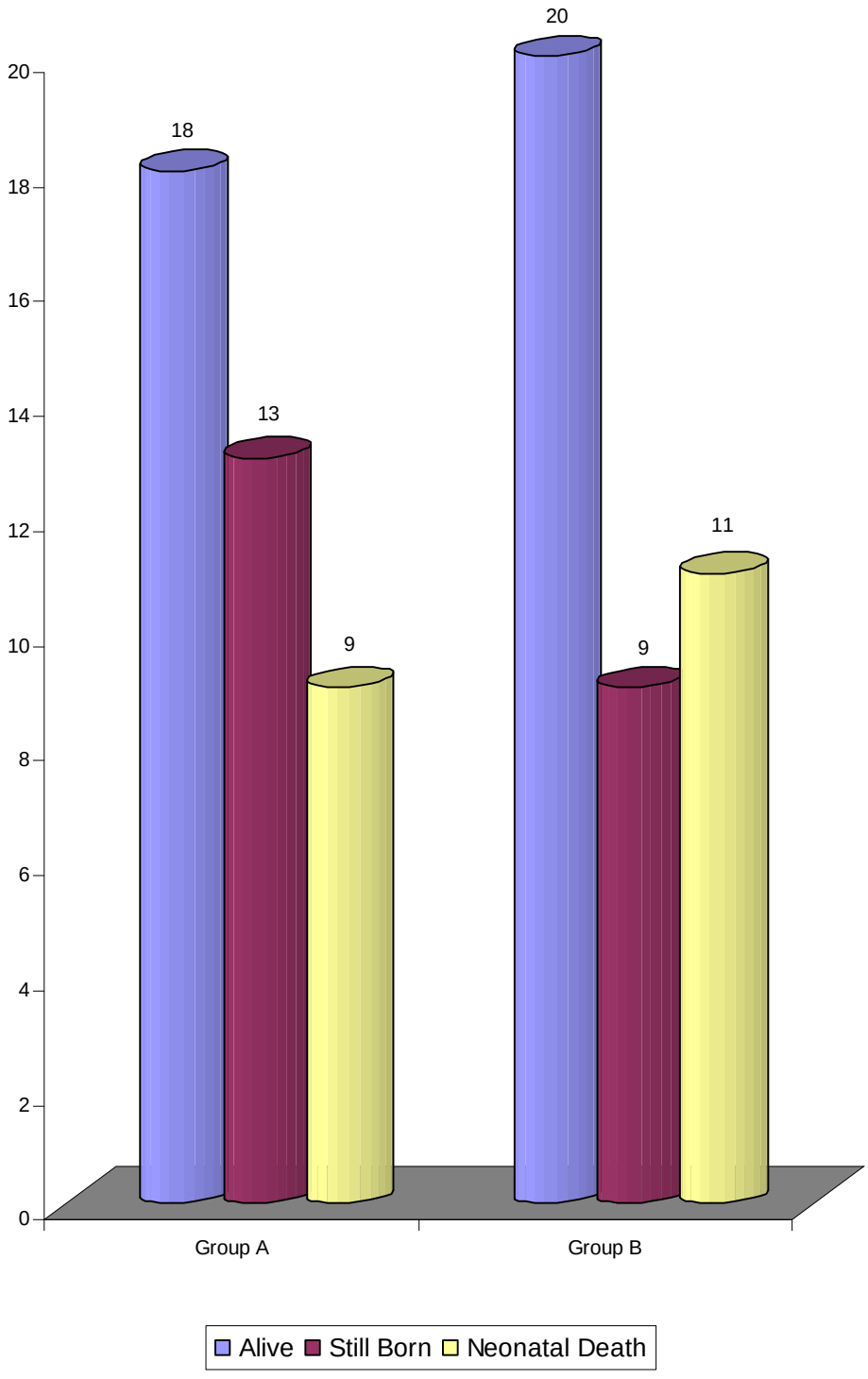


TABLE – 15

BIRTH WEIGHT OF BABIES IN THE TWO GROUPS

S. No.	Birth weight of Babies (in Kg.)	Group A	Group B
1.	Less than 1	01	01
2.	1 – 1.5	08	04
3.	1.6 – 2.5	21	27
4.	More than 2.5	10	08
5.	Mean	1.9356	1.5000
6.	Standard Deviation	0.59562	0.50315
7.	P Value	0.051	

The mean birth weight of the babies in Dhaka regimen group was 1.94 ± 0.59 kg and in Pritchard regimen group the mean birth weight was 1.5 ± 0.50 kg. The P value being 0.051 which is not significant.

FIG. 13

BIRTH WEIGHT OF BABIES IN THE TWO GROUPS

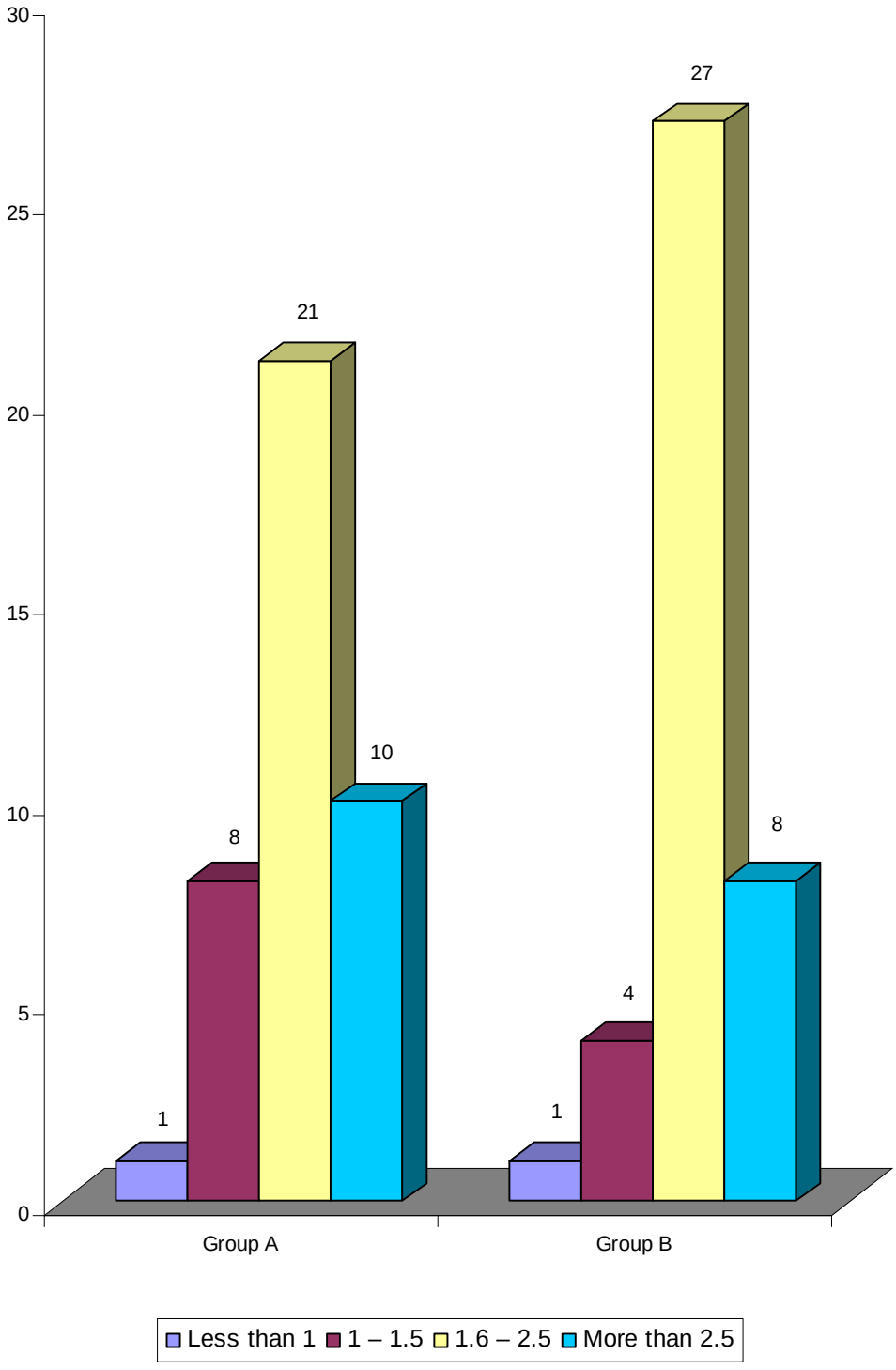


TABLE – 16

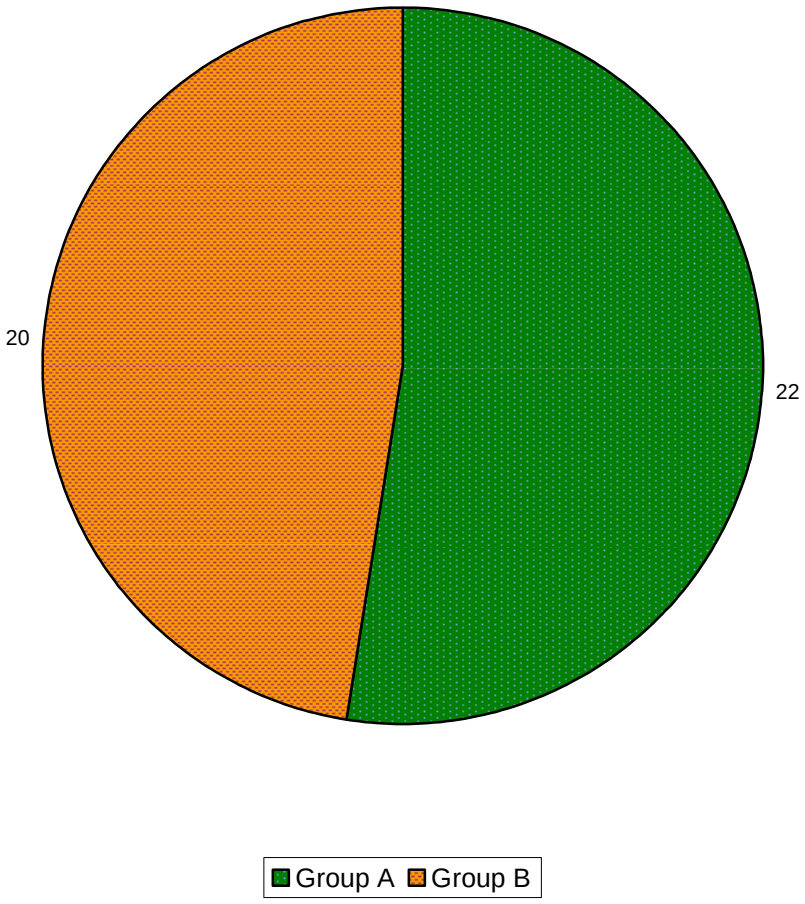
COMPARISON OF PERINATAL DEATHS IN BOTH GROUPS

	Total Births	Perinatal Deaths	
		No.	%
Group A	40	22	55
Group B	40	20	50

There were 22 perinatal deaths in group A out of 40 births and 20 perinatal deaths in group B out of 40 births.

FIG. 14

COMPARISON OF PERINATAL DEATHS IN BOTH GROUPS



DISCUSSION

DISCUSSION

Prevention of further seizures in eclampsia is associated with a reduction in adverse outcomes. Magnesium is an ideal drug, with rapid onset of action, a non sedative effect on mother and baby, a fairly wide safety margin and a readily available antidote in the form of calcium gluconate. The Collaborative Eclampsia Trial provided vital evidence that magnesium reduces the risk of recurrent seizures compared to other standard agents diazepam and phenytoin. Further more use of magnesium sulphate does not appear to be associated with detrimental effects on the neonate.

Evidence from computed tomography and magnetic resonance angiographic studies implicating cerebral vasospasm and ischemia in the genesis of eclampsia. Magnesium seems to reverse and ameliorate the effects of cerebral ischemia. There may also be a moderate inhibitory effects on cortical discharge with magnesium antagonizing the excitatory glutamate N-methyl aspartate receptor.

Falling serum calcium levels following administration of intra venous magnesium sulphate inhibit acetyl choline release at motor end plate.

In this study, a total of 10 gms of $MgSO_4$ as a loading does and 2.5 gms of $MgSO_4$ 4^{th} hourly as maintenance dose was used which was just over half the dose used by Pritchard regimen and in Collaborative Eclampsia Trial.

Age Distribution

A study in N.W.M. Hospital, Bombay in 1989 reveals that 40.5% were under 20 years, 56.8% were between 21 – 29 and 2.7% above 30 years. Lolkand *et al*. in his study (1997) found that 40.7% were under 20 years. In a study by Katz *et al.* (2000) in the sacred heart medical center USA the mean age of eclampsia was 22 years.

In this study, the mean age in Dhaka regimen group was 24.16 years and the mean age in Pritchard regimen group was 24.38 years.

Parity

In the study of Collaborative Eclampsia Trial Group (1995) 64% were primis. In the study by N.W.M. Hospital, Bombay (1989) 64.9% were primis. According to Mudhaliar over 75% were primis. In the

present study, in Dhaka regimen group 67.5% were primis and in Pritchard regimen group 60% were primis.

Gestational Age

In Collaborative Eclampsia Trial Group study (1995) 39.5% cases were less than 34 weeks and 25.5% cases were presented between 34 – 36 weeks and 33% cases were presented at term. In the present study mean gestational age in Dhaka regimen group was 32.93 weeks and in Pritchard regimen group it was 33.39 weeks.

Diastolic Blood Pressure

In Collaborative Eclampsia Trial Group study (1995) 53% had a diastolic blood pressure above or equal to 110 mm Hg. In the present study majority of patients had diastolic blood pressure between 100 – 110 mm Hg and in Dhaka regimen group it was 45% and in Pritchard regimen group it was 33%.

Recurrence rate of convulsions

The recurrence rate of convulsions after starting the regimen in Dommisse (1990) was 0%, in Collaborative Eclampsia Trial Group study (1995) was 5.7%, in PGI Chandigarh, it was 8.1%. In the present study,

5% of patients had recurrent convulsions in Dhaka regimen group and 2.5% of patients had recurrent convulsions in Pritchard regimen group.

Mode of induction

Alexander and colleagues (1999) reviewed 278 singleton liveborn infants weighing 750 – 1500 gm delivered by women with severe pre eclampsia in Parkland hospital. 50% were induced and 50% underwent caesarean delivery without labor. Induction was not successful in 35% of women in induced group. Similar results were reported by Nassar *et al*. (1918).

In the present study, in Dhaka regimen group 32.5% of patients were induced with syntocinon and 67.5% were induced with prostaglandin E_2 gel. Among them, 65% delivered vaginally and 35% underwent caesarean section.

In Pritchard regimen group, 60% were induced with syntocinon and 40% with prostaglandin E_2 gel. Among them, 67.5% delivered vaginally and 32.5% by caesarean section.

Perinatal Mortality

Perinatal deaths in Collaborative Eclampsia Trial Group (1995) with magnesium sulphate was 25%, with Diazepam it was 22% and with

Phenytoin it was 31%. In the present study, in Dhaka regimen group the total number of perinatal death was 22 and in Pritchard regimen group it was 20.

Maternal Morbidity and Mortality

The maternal mortality between 1991 – 1997 was approximately 6% in US were related to eclampsia. (Berg & Coworkers, 2003). The maternal mortality in Collaborative Eclampsia Trial Group 1995 with magnesium sulphate was 3.8%. In the present study, in both the Dhaka regimen and Pritchard regimen groups no maternal death occurred.

2 patients developed loss of knee jerk reflex with low dose Dhaka regimen group and 5 patients developed loss of knee jerk reflex in the Pritchard regimen group. In Pritchard regimen group 9 patients required dose deferral which is significantly higher than the Dhaka regimen group, in which only 3 patients required dose deferral due to loss of knee jerk reflex and oliguria. The mean serum magnesium level in Dhaka regimen was 4.03 mg/dl and in Pritchard regimen group it was 4.53 mg/dl. Both the values lie within the therapeutic serum magnesium level.

SUMMARY

SUMMARY

Variables	Dhaka regimen (Group A – 40)	Pritchard regimen (Group B – 40)
Recurrence of fits		
No. of cases	2	1
(%)	5%	2.5%
Mean Serum Magnesium Level	4.03 mg/dl	4.53 mg/dl
Mode of Delivery		
Vaginal	26	27
LSCS	14	13
Perinatal Outcome		
Live Born	18	20
Still Born	13	9
Neonatal death	9	11
Total Perinatal Deaths	22	20

§ In the present study, 40 Antepartum eclamptic patients were treated with lower dose Dhaka regimen and 40 Antepartum eclamptic patients were treated with Pritchard regimen of magnesium sulphate and the two groups were compared with respect to the safety and efficacy of treatment, complications, serum magnesium level, maternal and perinatal outcome.

- Recurrence of fits after starting the regimen was lower in both the magnesium sulphate regimen groups.
- Only 1 patient (2.5%) in the Pritchard regimen group had recurrence of fits and 2 patients (5%) had recurrence of fits in the Dhaka regimen group who were managed with an additional dose of 2 gm of intra venous $MgSO_4$.
- The mean serum magnesium level in Dhaka regimen group was 4.03 mg/dl and in Pritchard regimen group it was 4.53 mg/dl.
- 65% of patients delivered vaginally in Dhaka regimen group and 67.5% of patients delivered vaginally in Pritchard regimen group.
- LSCS was done for obstetric indications and for medical indications.
- 18 babies were alive in the Dhaka regimen group and 20 babies were alive in the Pritchard regimen group.
- The perinatal death in Dhaka regimen group was 22 (55%) and in Pritchard regimen group it was 20 (50%).
- There was no maternal mortality in both the groups and all mothers were discharged in good condition with no residual sequlae.
- The perinatal outcome does not differ significantly in both the groups.

CONCLUSION

CONCLUSION

Magnesium sulphate is the anti convulsant drug of choice in women with eclampsia. The low dose Dhaka regimen used for smaller women appears to control and prevent convulsions effectively. In the present study of Dhaka regimen, the serum magnesium levels remain below the toxic levels.

The present study provides further strong support for the routine use of magnesium sulphate for eclamptic convulsions. As long as there is adequate urinary output, clinical monitoring appears to be sufficient.

There is no difference in maternal mortality, perinatal mortality, maternal morbidity and cesarean section rates among the both magnesium sulphate regimen groups.

The induction delivery interval between the two groups does not differ significantly. The study clearly shows that Dhaka regimen is almost equivalent to Pritchard regimen, for the control of convulsions in eclampsia.

Proper antenatal care, improved socio economic status and intensive management will largely reduce the incidence of eclampsia

which is an important cause of maternal morbidity and mortality, perinatal morbidity and mortality.

In conclusion, seizures can be safely controlled in women with eclampsia with a lower dose of magnesium sulphate, with the advantage of a lower magnesium toxicity.

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PROFORMA

PROFORMA

NAME : AGE :

ADDRESS : IPNO :

OCCUPATION :

SOCIO ECONOMIC STATUS :

DATE & TIME OF ADMISSION :

DATE & TIME OF DELIVERY :

DATE OF DISCHARGE :

OBSTETRIC CODE :

LMP :

EDD :

GA :

BOOKED / UNBOOKED :

HISTORY OF PRESENTING ILLNESS:

Brought – Conscious /semiconscious/ Coma :

No. of convulsions before admission :

during admission :

Headache / Vomiting / Blurring of vision / Epigastric pain / Oliguria /

Pedal edema / Fever / fits

Labour pain / Bleeding PV / Draining PV

MENSTRUAL H/O

MARITAL H/O

OBSTETRIC H/O

Previous Obstetric Outcome

PAST H/O - HT / Epilepsy / Jaundice / DM / PIH /Eclampsia in previous pregnancy

PERSONAL H/O - Smoker / Non-smoker

FAMILY H/O - HT / Epilepsy / DM / Twins / Others

GENERAL EXAMINATION

Consciousness

Orientation

Pallor

Oedema – site and grading

Cyanosis

VITALS Temp PR BP RR

SYSTEMIC EXAMINATION

CVS

RS

CNS

DTR – Absent / Normal / Exaggerated

ABDOMEN

P / A EFW :

P / V Bishop Score :

STATUS OF PREGNANCY

Single / Multiple / Molar

PERIOD OF GESTATION

Term > 37 wks / Preterm 20-36 wks / < 20 wks

IN LABOUR - Yes / No – Duration

PLAN OF MODE OF DELIVERY

Induction / Accelaration

Vaginal / LSCS : Indication for LSCS

INVESTIGATION

URINE - Sugar / Albumin / Microscopy – Puscells, Casts

24 Hrs Urine protein / creatinine clearance

Complete Hemogram

Platelet count

BT

CT

CRT

Plasma Fibrinogen

Blood sugar

Blood Urea

Serum Creatinine

Serum Uric acid

Serum Electrolytes

Liver function test

Serum Magnesium

Peripheral smear study

USG

Fundus examination

MATERNAL OUTCOME

Mode of delivery – Vaginal / Instrumental / LSCS Time of delivery

$MgSo_4$ Regimen given

No. of recurrent convulsions after receiving $MgSo_4$

Did the mother show signs of toxicity

Maternal complications during hospital stay

Condition of the mother at discharge – Healthy / Residual Sequlae / died

FETAL OUTCOME

Still born / Alive - Sex

Apgar score - 1min & 5 mins

Weight - Kg

Congenital abnormality if any -

Condition of child at discharge - Healthy / Residual sequlae / died

Cause of Neonatal death

MASTER CHART

S. No.	Name	Age	IP No.	OBST Code	Book Status	GA in WKS	No. of fits BEF ADM	Level of Conscious	BP (mm Hg)		Reg. Group	S. Mg mg/dl	MOI	MOD	Adm. Del. Int. (Hrs.)	Maternal Outcome				Perinatal Outcome	
									Syst.	Dias						Seizure recurrence	Signs of $MgSO_4$ toxicity	Dose deferral	Condition at discharge	Bwt	Condition
1.	Akilandam	26	23212	$G_2P_1L_1$	2	34	4	2	170	110	A	4.2	0	2	0.5	-	-	-	1	2.1	1
2.	Mayil	22	24228	$G_2P_1L_1$	1	32	3	2	160	100	A	4	3	1	6	-	-	-	1	1.8	2
3.	Juliet Regina	35	24301	$G_4P_3L_3$	2	30	1	2	160	120	B	4.6	3	1	7	-	+	3	1	1.2	2
4.	Sathya Priya	24	25363	Primi	1	36	2	2	180	110	A	3.8	1	2	2.4	-	-	-	1	2.5	1
5.	Rathna	25	25488	Primi	1	32	2	1	170	110	B	5.2	3	1	5	-	-	-	1	2.1	1
6.	Pappathi	29	26040	$G_2P_1L_1$	2	34	3	2	160	110	B	5	1	1	6.2	-	-	-	1	2.2	1
7.	Semmina	24	27151	Primi	2	28	7	2	150	100	A	3.2	1	1	8	-	-	-	1	0.9	2
8.	Karpagam	37	27426	Primi	1	36	5	2	140	110	B	4.8	1	2	8	-	+	1	1	2.7	1
9.	Mala	32	27575	Primi	2	38	3	2	180	120	B	3	3	2	1.5	+	-	-	1	2.7	1
10.	Fairose	27	28279	$G_3P_2L_1$	2	36	2	2	190	110	A	4.4	3	1	3	-	-	-	1	2	3
11.	Muthulaxmi	25	29672	$G_2P_1L_0$	1	32	4	2	160	100	A	4	1	1	8	-	-	-	1	1.9	3
12.	Rebamaxy	31	30350	$G_3P_2L_2$	1	34	3	2	150	110	A	3.7	3	1	4.5	-	-	-	1	2.1	1
13.	Palaniammal	20	30495	Primi	2	32	1	1	140	90	B	4.6	1	1	8.4	-	-	-	1	1.8	1
14.	Sathya	23	30632	$G_2P_1L_1$	2	36	3	2	160	100	B	4.7	3	2	7.3	-	-	-	1	2	1
15.	Uma	24	31562	$G_3P_2L_2$	2	34	2	2	160	110	B	5.2	3	1	4	-	-	-	1	2	3
16.	Parasakthi	29	32985	$G_2P_1L_1$	2	24	3	2	150	110	A	4	1	1	11	-	-	-	1	0.8	2
17.	Sangeetha	19	34196	Primi	1	28	2	1	150	90	A	4.2	1	1	9	-	-	-	1	1	2
18.	Revathi	20	34906	Primi	2	36	3	2	140	100	B	4.8	3	1	6	-	-	-	1	2.7	1
19.	Iyyammal	21	36519	Primi	1	38	4	1	130	90	B	5.4	1	2	9	-	-	-	1	2.6	1
20.	Maheswari	20	36934	Primi	1	36	6	2	160	110	A	4.8	1	2	8	-	-	-	1	2.5	1

S. No.	Name	Age	IP No.	OBST Code	Book Status	GA in WKS	No. of fits BEF ADM	Level of Conscious	BP (mm Hg)		Reg. Group	S. Mg mg/dl	MOI	MOD	Adm. Del. Int. (Hrs.)	Maternal Outcome				Perinatal Outcome	
									Syst.	Dias						Seizure recurrence	Signs of $MgSO_4$ toxicity	Dose deferral	Condition at discharge	Bwt	Condition
21.	Sumathi	27	38770	$G_3P_2L_1$	1	32	5	2	170	120	B	5.3	3	1	4	-	-	-	1	2.2	3
22.	Selvarani	25	39464	$G_2P_1L_1$	2	34	4	1	150	100	A	5.2	3	2	6	-	-	-	1	2	3
23.	Manjula	25	40993	Primi	2	34	3	2	150	110	B	5.8	1	1	8	-	-	-	1	1.8	2
24.	Padma	19	42098	Primi	1	30	2	1	140	90	A	4	1	1	11	-	-	-	1	1.2	2
25.	Lalitha	26	42797	Primi	1	36	2	2	180	110	B	4.8	1	2	7	-	+	+	1	1.9	1
26.	Lakshmi	20	44163	Primi	1	32	8	2	170	110	A	4.2	1	1	8	-	-	-	1	1.5	2
27.	Hairunisa	21	46058	Primi	1	34	3	2	160	120	A	3.8	3	1	5	-	-	-	1	1.5	3
28.	Kokila	24	49630	$G_2P_1L_1$	1	38	1	1	190	100	A	4	1	2	10	-	-	-	1	2.9	1
29.	Sellarani	22	49942	$G_2P_1L_0$	1	38	4	2	180	100	A	4	1	2	10	-	-	-	1	2.9	1
30.	Kalaiselvi	21	50442	Primi	1	30	1	1	130	100	B	4.6	1	1	7.5	-	-	-	1	1.7	3
31.	Tamilselvi	20	57376	Primi	1	34	3	2	160	110	B	5	1	1	0.3	-	-	-	7	2.1	1
32.	Pappathi	27	51548	$G_2P_1L_1$	2	32	1	1	170	110	A	4.2	1	1	4	-	-	-	1	1.8	3
33.	Poomani	21	51872	Primi	1	36	4	2	180	120	B	4.8	1	2	0.5	-	-	-	1	2.2	1
34.	Thilagavathi	22	52004	Primi	2	34	2	1	150	100	A	3.6	1	1	6	-	-	-	1	2	1
35.	Lalitha	25	53134	$G_3P_2L_2$	1	36	5	2	170	120	B	5.4	3	2	7	-	-	-	1	2.5	1
36.	Rajeswari	19	53868	Primi	1	30	3	2	160	100	A	4.4	1	1	10	-	-	-	1	1.7	2
37.	Rahmath	20	54551	Primi	1	36	2	2	170	110	A	4.6	3	1	1.2	-	-	-	1	2.3	1
38.	Banupriya	24	55410	Primi	1	38	6	2	160	100	B	6	3	2	2.5	-	+	+	1	2.6	1
39.	Vellammal	30	57986	$G_3P_2L_2$	1	34	5	1	190	120	B	5.8	3	1	1	-	-	-	1	3	1
40.	Aruldevi	27	1794	$G_2P_1L_1$	2	34	6	2	170	110	A	4.8	1	1	6	-	+	+	1	2.4	3

S. No.	Name	Age	IP No.	OBST Code	Book Status	GA in WKS	No. of fits BEF ADM	Level of Conscious	BP (mm Hg)		Reg. Group	S. Mg mg/dl	MOI	MOD	Adm. Del. Int. (Hrs.)	Maternal Outcome				Perinatal Outcome	
									Syst.	Dias						Seizure recurrence	Signs of $MgSO_4$ toxicity	Dose deferral	Condition at discharge	Bwt	Condition
41.	Sathya	23	1892	Primi	2	32	3	2	160	90	B	5.6	1	1	13	-	+	+	1	2	3
42.	Suguna	26	2126	Primi	1	30	2	2	130	100	B	5.2	3	1	2.5	-	-	-	1	1.6	3
43.	Thangam	20	1577	Primi	1	36	4	2	160	110	A	3.8	3	2	2	-	-	-	1	2.4	1
44.	Lakshmi	22	2588	Primi	2	28	2	2	150	100	A	3.6	1	1	12	-	-	-	1	1	2
45.	Kasambu	25	4207	$G_2P_1L_1$	2	34	1	1	200	120	B	4.8	3	2	3	-	-	-	1	2	3
46.	Usha	23	8330	Primi	1	36	6	2	170	110	B	4.4	1	1	10	-	-	-	1	2.4	1
47.	Valarmathi	25	9502	Primi	1	34	3	2	140	100	A	4.2	3	1	6	-	-	-	1	2.1	3
48.	Amudha	28	10310	$G_4P_3L_2$	2	33	2	2	190	120	A	4.6	3	1	2	-	+	+	1	2	2
49.	Lakshmi	27	13512	Primi	2	28	2	2	180	110	A	4	1	2	1.5	-	-	-	1	0.9	2
50.	Rathnammal	25	14436	Primi	1	24	4	2	170	120	A	4.4	1	1	10	-	-	-	1	0.7	2
51.	Parimala	24	15069	Primi	1	32	3	2	160	110	B	4.2	1	1	9	-	-	-	1	1.6	2
52.	Nirmala	27	15512	Primi	2	34	1	1	180	120	B	4.6	1	1	7	-	+	+	1	1.8	3
53.	Revathi	21	15642	Primi	1	38	3	2	190	120	B	4.8	1	2	2.5	-	-	-	1	3	1
54.	Chellathaye	22	16372	Primi	1	32	2	1	170	120	A	4.2	1	2	7	+	-	-	1	2	3
55.	Rajeshwari	19	16763	Primi	1	34	3	2	150	110	A	3.8	1	1	10	-	-	-	1	2.1	1
56.	Maha	25	18185	$G_4P_3L_3$	2	28	5	2	160	120	B	5.2	3	1	4	-	-	-	1	1.5	2
57.	Parameshwari	25	15388	Primi	1	26	1	1	180	100	A	4	1	1	11	-	-	-	1	0.8	2
58.	Vanitha	22	20376	Primi	2	32	1	2	190	120	A	3.6	1	2	8	-	-	-	1	1.6	2
59.	Kalaiselvi	35	21636	$G_5P_2L_2A_2$	2	32	3	2	190	130	B	5	1	1	7	-	-	-	1	1.5	2
60.	Radhika	30	22733	Primi	1	26	2	7	200	110	B	5.2	1	1	12	-	-	-	1	0.6	2

S. No.	Name	Age	IP No.	OBST Code	Book Status	GA in WKS	No. of fits BEF ADM	Level of Conscious	BP (mm Hg)		Reg. Group	S. Mg mg/dl	MOI	MOD	Adm. Del. Int. (Hrs.)	Maternal Outcome				Perinatal Outcome	
									Syst.	Dias						Seizure recurrence	Signs of $MgSO_4$ toxicity	Dose deferral	Condition at discharge	Bwt	Condition
61.	Suganya	20	24121	Primi	2	30	2	2	160	100	B	5	1	1	5	-	-	-	1	1.5	2
62.	Selvamani	19	27951	Primi	1	26	4	2	150	120	A	4.2	1	1	11	-	+	+	1	0.75	2
63.	Vijaya	27	29525	Primi	1	28	1	2	160	110	A	4.4	1	1	10.2	-	-	-	1	1	2
64.	Neelavathi	20	31470	Primi	1	32	3	2	170	110	B	4.8	1	1	8	-	-	-	1	2	1
65.	Priya	18	31583	G_2A_1	2	34	3	2	180	100	B	5.2	1	2	3.4	-	-	-	1	2.3	1
66.	Vijayarani	20	31699	Primi	1	36	2	1	170	110	A	4.4	3	2	2.1	-	-	-	1	2.8	1
67.	Akila	27	32133	$G_2P_1L_1$	1	34	8	2	160	120	B	5	1	1	8	-	+	+	1	2	3
68.	Manjula	25	32393	Primi	1	32	3	2	150	100	B	4.8	1	1	9	-	-	-	1	1.9	3
69.	Saradha	25	33157	$G_4P_3L_1$	1	34	4	2	170	120	A	4.2	3	1	4	-	-	-	1	1.5	2
70.	Selvi	35	35255	$G_2P_1L_1$	2	32	4	2	170	100	B	5.2	3	1	5	-	-	-	1	1.6	2
71.	Annakamu	24	36210	Primi	1	28	7	2	170	110	A	4	1	1	11	-	-	-	1	1.25	2
72.	Sangeetha	27	37262	$G_2P_1L_0$	1	36	4	2	180	120	B	4.8	3	2	3.5	-	-	-	1	2	3
73.	Revathi	20	37290	Primi	1	30	1	2	180	130	A	3.6	1	1	10	-	-	-	1	2	3
74.	Rosemary	20	37321	Primi	1	34	4	2	160	120	A	3.4	1	2	7.3	-	-	-	1	2.1	1
75.	Veerayee	19	38100	Primi	1	36	2	1	170	110	A	3.2	3	2	0.5	-	-	-	1	2.7	1
76.	Noorjahan	23	39206	$G_3P_2L_2$	1	38	5	2	150	190	B	5.4	3	1	3	-	+	+	1	3.1	1
77.	Muthukani	20	39329	$G_2P_1L_1$	2	36	3	2	190	110	B	5.2	1	2	6.3	-	-	-	1	2.3	1
78.	Kavitha	24	39411	Primi	1	32	4	2	160	110	B	5.6	1	1	9	-	+	+	1	1.8	2
79.	Sarasu	22	39452	$G_2P_1L_1$	1	34	2	1	170	100	B	5	1	1	3.3	-	-	-	1	2.1	3
80.	Kamatchi	26	39591	Primi	1	36	3	2	170	120	A	4.2	1	2	8	-	-	-	1	2.75	1

ABBREVIATIONS

OBST CODE	-	Obstetric Code
BOOK STATUS	-	Booking Status
1	-	Booked
2	-	Unbooked
GA	-	Gestational Age in weeks
No. of fits BEF ADM	-	Number of fits before Admission
Level of Conscious		
1	-	Conscious
2	-	Semiconscious
BP	-	Blood Pressure
Syst.	-	Systolic
Dias.	-	Diastolic
Reg. Group	-	Regimen Group
A	-	Dhaka regimen
B	-	Pritchard regimen
S. Mg	-	Serum Magnesium
MOI	-	Mode of Induction
0	-	No induction
1	-	Prostaglandin E_2 gel
2	-	Oxytocin
MOD	-	Mode of Delivery
1	-	Vaginal Delivery
2	-	LSCS
ADI	-	Admission to Delivery Interval (in hours)
Condition of Mother at Discharge		
1	-	Alive
2	-	Residual Sequlae
3	-	Dead
Condition of the baby at Discharge		
1	-	Alive
2	-	Still born
3	-	Neonatal death